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ACETYLCHOLINE IN THE TREATMENT OF ACUTE RETROBULBAR NEURITIS

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Owing to the fact that sodium nitrite administered intravenously and amyl nitrite given by inhalation have both been found to be very effective in aborting attacks of acute retrobulbar neuritis,[1] with resulting rapid improvement in vision, it was deemed advisable to study the action of acetylcholine, a powerful but little used vasodilator, in this interesting disease.

In 1931 de Saint-Martin [2] reported a case of retrobulbar neuritis in which he thought the condition was improved after the use of acetylcholine. A woman of 31 had noticed gradual failure in vision, which began six weeks after the birth of a child. Five months later vision was found to be 6/10 in the right eye and perception of light in the left eye. The visual fields were contracted, and there was probably a central scotoma in each eye. The fundi were normal. A submucous resection of the nasal septum was done, and subcutaneous injections of atropine sulfate (1 mg.) were given every other day. Vision improved to 8/10 in the right eye in eight days and to 4/10 in the left eye in one month. Three months later vision was 7/10 in the right eye and 4/10 in the left eye. At this time one of the turbinate bones was removed. Three weeks after this second nasal operation vision was unchanged; so alternating injections of acetylcholine chloride and atropine were given for six days, after which the atropine was discontinued. Six weeks later, after twelve injections of acetylcholine had been given, vision was 10/10 in the right eye and 8/10 in the left. This improvement was maintained for two months, during which time twelve more injections of acetylcholine were given. This case was complicated by the fact that

From the Herman Knapp Memorial Eye Hospital.

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1. Duggan, W. F.: Use of Vasodilators in Retrobulbar Neuritis, Arch. Ophth. **16**:380 (Sept.) 1936.

2. de Saint-Martin: Concerning the Efficiency and Diagnostic Value of Vasodilators, Especially Acetylcholine, in Retinal Angiospasms, Ann. d'ocul. **168**: 102 (Feb.) 1931.

Saint-Martin did not limit himself to one type of treatment. However, his explanation is that the improvement which followed the first nasal operation was due to the use of atropine subcutaneously, since no improvement occurred after the second operation until vasodilator therapy was instituted.

In 1932 Villaret, Justin-Besançon, Schiff-Wertheimer and Gallois [3] stated that acetylcholine should be of value in the treatment of retrobulbar neuritis but that they had no personal experience of its value in such cases. However, both Bonnefon [4] and Orr [5] have reported good results following its use in chronic retrobulbar neuritis (toxic amblyopia).

CHEMISTRY AND PHYSIOLOGY OF ACETYLCHOLINE

In 1934 Alles [6] reviewed the physiology of acetylcholine and other derivatives of choline, and in 1935 Gaddum [7] wrote a comprehensive survey of the biochemical properties of the same substances. Both these articles have been used in preparing this section of this report.

Acetylcholine may be regarded as the product of a reaction between choline and acetylchloride. The presence of the acetyl group in the choline molecule increases the pharmacologic activity of choline at least a thousandfold. Thus, Burn [8] found that the intravenous injection of 0.001 mg. of acetylcholine into a cat caused an immediate fall in the systolic blood pressure from 100 to 35 mm. of mercury and that the blood pressure returned to normal in approximately ninety seconds. (This amount of acetylcholine corresponds to a concentration of approximately 1 : 100,000,000.) With this fall in the blood pressure it was also shown that there was a marked dilatation of the arterioles in the limbs of the cat.

Although acetylcholine is such a powerful vasodilator, it is rapidly hydrolyzed and inactivated in the presence of extracts of blood or of tissue by a specific choline esterase which has been isolated. At 40 C. acetylcholine is almost completely destroyed by human blood within fifteen seconds.

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3. Villaret, M.; Justin-Besançon, L.; Schiff-Wertheimer and Gallois, J.: The Esters of Choline in Ophthalmology, Arch. d'opht. **49**:129 (March) 1932.

4. Bonnefon: The Value of Acetylcholine in the Treatment of Toxic Amblyopia, Prat. méd. franç. **12**:65 (Feb.) (B) 1931.

5. Orr, H. Campbell: Acetylcholine in Tobacco Amblyopia, Brit. M. J. **2**: 69 (July 11) 1936.

6. Alles, G. A.: The Physiological Significance of Choline Derivatives, Physiol. Rev. **14**:276 (April) 1934.

7. Gaddum, J. H.: Choline and Allied Substances, in Luck, J. M.: Annual Review of Biochemistry, Stanford University, Calif., Stanford University Press, 1935, vol. 4, p. 311.

8. Burn, quoted by Evans, C. L.: Recent Advances in Physiology, ed. 4, Philadelphia, P. Blakiston's Son & Co., 1930.

Physostigmine has a powerful inhibitory action on choline esterase, although this inactivation is slow and reversible. A probable theory of this inhibition has been evolved by Stedman and Stedman,[9] who suggested that physostigmine, which is itself a stable ester, acts by combining with the esterase in the same way and by the same mechanism as the choline esters do, thus blocking the enzyme. Moreover, urethanes inhibit choline esterase, and since physostigmine has a urethane group it is probable, according to Krause,[10] that it is the urethane group of physostigmine which combines with the choline esterase.

At present, pharmacologic methods are used in estimating the amount of acetylcholine in tissues; 0.000001 mg. can be detected qualitatively, and 0.001 mg. can be estimated quantitatively, with an error not greater than 10 per cent. The most specific and sensitive tissue for the detection of acetylcholine is the longitudinal muscle from the anterior end of the dorsum of a leech (Hirudo medicinalis) suspended in a salt solution which contains physostigmine. Less sensitive tests include the action of acetylcholine on the isolated auricle of the rabbit's heart, on the frog's heart, on the small intestine of the mouse or of the rabbit, on the voluntary muscles of the frog and on the blood pressure of the cat or its effect on a cat's voluntary muscle which has been sensitized by previous section of the motor nerve.

It is extremely probable that acetylcholine is liberated in the tissues by the action of certain nerves (especially the parasympathetic nerves), just as an epinephrine-like substance (sympathin) is liberated by stimulation of the sympathetic nerves, and that the effects obtained by stimulating autonomic nerves is due to the liberation of acetylcholine or sympathin. Conversely, the results following the injection of acetylcholine simulate the results obtained by stimulating a parasympathetic nerve (just as the results following the injection of epinephrine hydrochloride simulate those caused by the stimulation of sympathetic nerves), only the action is fleeting and evanescent except in the presence of physostigmine. These facts are the basis for the theory of the humoral transmission of nervous impulses, particularly with reference to the autonomic nervous system. At present there is no purely chemical evidence identifying the substance liberated by the parasympathetic nerves as acetylcholine, but a large mass of circumstantial evidence has been accumulated by the work of Loewi, Dale, Gaddum, Chang, Feldberg, Magnus and many others.[11]

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9. Stedman and Stedman, quoted by Gaddum.[7]

10. Krause, A. C.: The Biochemistry of the Eye, Monograph 2, Baltimore, Md., Johns Hopkins Press, 1934.

11. Footnotes 6 and 7.

RÔLE OF ACETYLCHOLINE IN OCULAR MECHANISMS

In 1931 Englehart [12a] reported that after stimulation of the third nerve the vitreous of a rabbit's eye which had been treated previously with physostigmine contained a substance which slowed the heart of a cold-blooded animal. Normal vitreous had no such effect. Moreover, when both eyes were treated with physostigmine and one eye was exposed to light the vitreous of this eye was rich in this substance (*Vagusstoff*, or acetylcholine) while the eye kept in darkness contained little or none of this substance. (Rabbits do not have a consensual light reflex.) Englehart expressed the belief that such a reaction is comparable to the effect of the vagus on the heart and that it is a humoral mechanism. Also, when the iris and ciliary body were isolated from the oculomotor nerve they contained none of this reactive substance (acetylcholine), but when the third nerve was intact and was stimulated electrically this substance could be detected in the ciliary body and iris.[12b] This author later reported [12c] finding a substance in the aqueous which was similar in all respects to acetylcholine when experiments similar to those just described were carried out, it being necessary, of course, to use physostigmine first to prevent the rapid disappearance of the substance.

In 1932 Velhagen [13a] reported finding a substance similar to acetylcholine in alcoholic extracts of the uvea and retina as confirmed by its ability to contract the pupil and lower the intra-ocular tension. In 1933 he showed [13b] that acetylcholine was able to constrict the isolated sphincter iridis in a dilution as high as 1 : 4,000,000, the effect being abolished by a previous application of atropine. In 1935, using the dorsal muscle of a leech which had been sensitized by the previous application of physostigmine, he demonstrated the presence of acetylcholine in the retina and uveal tract.[13c]

According to Krause,[10] both choline and acetylcholine are found in the normal moist bovine retina. It is assumed that they are formed in the retina from an unknown precursor as a step in the photochemical processes of vision.

In 1930 Duke-Elder [14a] reported that the extra-ocular muscles contract in vivo following the intravenous injection of acetylcholine and

12. Englehart, E.: (*a*) The Mode of Oculomotor Stimulation, Klin. Wchnschr. **10**:26 (Jan. 3); (*b*) 215 (Jan. 31) 1931; (*c*) The Humoral Mechanism of Stimulation of the Third Nerve, Arch. f. d. ges. Physiol. **227**:220, 1931.

13. Velhagen, K.: (*a*) Intra-Ocular Substances Like Acetylcholine, Klin. Monatsbl. f. Augenh. **88**:846, 1932; abstr., Arch. Ophth. **9**:493 (March) 1933: (*b*) Experiments on Isolated Iris Muscles in Pharmacologic Research, Arch. f. Augenh. **107**:172, 1933; (*c*) Further Experiments on Active Principles in Ocular Tissues, ibid. **109**:195, 1935.

14. Duke-Elder, W. S.: (*a*) New Observations on the Physiology of the Extra-Ocular Muscles, Tr. Ophth. Soc. U. Kingdom **50**:181, 1930; (*b*) Text-Book of Ophthalmology, London, Henry Kimpton, 1932, vol. 1

in vitro [14b] when this substance is added to a muscle bath containing physostigmine. This action is unaffected by atropine, increased by epinephrine and abolished by both nicotine and curare. A similar reaction occurs in the normal striated muscles of species below the mammals and in the muscles of fetal mammals but in the voluntary muscles of fully developed mammals only after they have been deprived of their motor nerve supply. "At present, the significance of this reaction is not clear. It would seem that these muscles (extra-ocular) are able to avail themselves of an archaic and primitive mechanism which the other voluntary muscles have lost and which is analogous to that evident in the involuntary muscles" (Duke-Elder [14b]).

This brief summary will serve as an introduction to the use of acetylcholine in the treatment of acute retrobulbar neuritis. It must be emphasized that its use in this condition is dependent only on its vasodilating property. Moreover, the action of acetylcholine as a vasodilator is probably very transient, owing to its rapid inactivation by the choline esterase present in the blood and tissues. This characteristic, while probably decreasing its efficiency, adds a factor of safety to its use.

REPORT OF CASES

The two cases of acute retrobulbar neuritis which were the occasion for the present article are described briefly.

CASE 1.—C. P., a man of 31, was seen on Jan. 8, 1934, with the complaint of blurred vision in the right eye, which had been present for approximately two months. A tonsillectomy on Nov. 17, 1933, had been followed by a slight temporary improvement in vision; on December 18 vision was 20/30. There was no history of a recent cold in the head, and the patient did not use tobacco or alcohol.

Vision in the right eye (corrected) was 20/50 on Jan. 8, 1934, and 20/40 on January 12. The fundus showed hyperemia and nasal blurring of the disk, with engorgement of the retinal veins. On the tangent screen there was an oval scotoma 2 by 5 degrees in size just temporal to the fixation point for a 1 mm. white test object at a distance of 1 meter, and a 5 mm. red test object at a distance of 1 meter was identified only in a crescentic area in the nasal field. Vision in the left eye (corrected) varied between 20/30 and 20/20 on several occasions; the field was normal, and the fundus was similar to that of the right eye except that the retinal veins were not engorged. Both eyes had a high degree of compound hyperopic astigmatism. The Wassermann reaction of the blood was negative; there were no signs of multiple sclerosis, and the sinuses were normal on clinical examination.

This patient received three intramuscular injections of acetylcholine (0.1 Gm.) between January 12 and 19. On the tenth day after this treatment was begun (January 21) vision in the right eye was 20/20—, the scotoma was barely perceptible and the field for red was markedly improved. The fundus showed no change. This improvement was maintained for the next ten days, during which time two more injections of acetylcholine were given.

On Nov. 13, 1936, Dr. W. F. C. Steinbugler informed me that vision in the right eye was 20/20— and that the patient was not aware of any improvement in

vision until sphenoid sinusitis (diagnosed by the roentgen rays) had been treated. He received this treatment some time after Jan. 31, 1934. His failure to attribute any improvement to the injections of acetylcholine was probably due to the fact that he still had a small scotoma when he was last seen; he received injections every three days instead of daily as in case 2.

CASE 2.—R. C., a married woman of 21, was seen first on Dec. 2, 1935, with the history that four days before she had noticed marked blurring of the left vision on awakening. As she had been nursing a baby for three months the case might be classified as one of retrobular neuritis associated with lactation. However, a severe cold in the head developed on the day after her vision failed, which might also have been an etiologic factor.

Vision in the left eye was 8/200; the media were clear, and examination of the fundus revealed some blurring of the disk, with engorgement of the retinal veins. There was a dense scotoma about 30 degrees in diameter, including both the blindspot and the fixation point, for a 5 mm. white test object at a distance of 1 meter. A 10 mm. red test object at a distance of 1 meter was not identified in any part of the field. Vision of the right eye (corrected) was 20/25, and the field was normal. Both eyes were highly astigmatic.

Daily intramuscular injections of acetylcholine were instituted at once. On the fifth day after they were begun (December 6) immediately after the fifth injection vision in the left eye was 20/25 +. A small cecocentral scotoma for a 1 mm. white test object at a distance of 1 meter was present, and a 10 mm. red test object at a distance of 1 meter was perceived normally, except in the region of the cecocentral scotoma.

One month later vision was unchanged, and the field was completely normal. It was impossible to persuade this patient to have either a neurologic or a rhinologic examination.

The cases of eight patients suffering from attacks of acute retrobulbar neuritis who were treated with intravenous injections of sodium nitrite or with inhalations of amyl nitrite (one case) have been reported previously.[1] One patient was not improved; the other seven patients attained their best final vision in an average time of eight and one-half days.[15] The two patients treated with acetylcholine obtained their best final vision in ten days and five days, respectively, the average time being seven and one-half days. Although only two cases are presented in this report, it must be evident that all these patients were treated only with vasodilators, so the results obtained in the patients treated with acetylcholine confirm the work done earlier with sodium nitrite and amyl nitrite.

SUMMARY

The chemistry and physiology of acetylcholine have been briefly reviewed.

The rôle of acetylcholine as a fundamental factor in the mechanism of the action of the parasympathetic (cholinergic) nerves has been outlined.

15. The time of improvement includes both the day on which treatment was begun and the first day that the best final vision was attained.

The relation of acetylcholine to normal ocular mechanisms has been briefly reviewed.

In the treatment of acute retrobulbar neuritis the vasodilating property of acetylcholine is of paramount importance.

CONCLUSIONS

Two patients with acute retrobulbar neuritis were treated only with acetylcholine injected intramuscularly; vision improved from 20/40 to 20/20— in ten days in the first case and from 8/200 to 20/25 in five days in the second case.

These results compare favorably with those obtained in eight other cases in which only intravenous injections of sodium nitrite, inhalations of amyl nitrite or a combination of the two was used.

The concept that acute retrobulbar neuritis is due to a vascular spasm in the optic nerve is relatively new and is based only on therapeutic results. As the treatment is simple for both the physician and the patient, it deserves further investigation by ophthalmologists before its actual value can be definitely evaluated.

DISCUSSION

DR. WILLIAM F. C. STEINBUGLER: I had occasion to see the patient first reported on and observed him for a period of four years before he came to me in November 1933 with the history of blurred vision of the right eye of short duration. I then made a diagnosis of retrobulbar neuritis and referred the patient to a rhinologist, who promptly removed his tonsils. After that the patient was transferred to the Herman Knapp Memorial Eye Hospital, so that I cannot report the effect that the injections had on his vision. At Dr. Duggan's request I examined the patient several days ago; vision then was 20/20 in the right eye and 20/30 in the left eye. The patient is positive that he obtained no lasting improvement until he had treatment for his sphenoid sinus, some time early in 1934.

DR. S. A. AGATSTON: I am puzzled about the vascular aspect of retrobulbar neuritis, because there is no definite proof that retrobulbar neuritis is caused by vascular spasm. Dr. Duggan seems to think there is a spasm of an artery in the optic nerve and stated in both of his case reports that the first discoverable sign was engorgement of the retinal veins. That in itself would indicate that there could not have been a spasm of the retinal artery, because in spasm of the retinal artery there is no engorgement of the veins. In retrobulbar neuritis or in any condition that causes a great deal of congestion of the optic nerve there is pressure on the vessels, the first effect being engorgement of the veins and the second effect, compression of the arteries, rather than spasm of the arteries. I do not deny the possibility that the vasodilators may effect improvement of the circulation mechanically. My only objection to the theory is that I do not think that the changes in the vessels can be caused by retinal arterial spasm.

TRAUMATIC GLAUCOMA

AN ANATOMIC AND CLINICAL STUDY

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I. ANATOMIC PART

Anatomic descriptions of traumatic glaucoma are rare, as only two cases have been published (Garnier [Garnye],[1] 1891; Morax, 1922). To this small number a few other cases may be added in which the eyeball had to be removed after contusion.

To exclude the possibility of intra-ocular infection clouding the picture, cases in which there was a complete rupture of the sclera or a perforating wound were not included in this study.

CASES REPORTED IN THE LITERATURE

CASE 1 (Garnier, 1891).—The globe of a boy aged 15 was removed fifteen days after an accident.

The sclera showed staphyloma medially and superiorly near the equator. The anterior drainage channels were normal. The pectinate ligament showed open clefts. Schlemm's canal was wide and open. The iris was hyperemic. Otherwise there were no pathologic changes; no displacement, no synechiae and no sclerosis. Examination of the lens revealed no dislocation and no cataract. The zonule was torn in one place. The ciliary body showed edema; the processes were larger and branched more markedly than normally. The ciliary epithelium was swollen and depigmented in many places by maceration; the bundles of muscles were pressed apart by fluid, and many muscle fibers were turbid and opaque. At the level of the tear of the zonule the radial muscle fibers were torn over a distance of about 1 mm. The large circular artery had also been torn and had thrombosed; the hemorrhage caused by this tear had been resorbed. The choroid showed marked hyperemia and edema everywhere; the suprachoroid was filled with fluid, especially at the equator, so that it had attained from five to six times its normal width. Examination of the sclera showed that the lymph channels in the sheaths of the blood vessels and the nerves were filled with pigment cells which had a lighter color than the choroidal cells; these cells were derived from the choroid and had been loosened by maceration. The optic nerve showed a mass of young connective tissue anterior to the optic papilla, perhaps a remnant of a previous hemorrhage.

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1. The spelling "Garyne" rather than "Garnier" is given first in the bibliography, since the article cited appeared in a Russian journal and the form "Garnye" is used in the Russian literature and in references to it in standard indexes.

In this case the pectinate ligament, Schlemm's canal, the angle of the anterior chamber and the iris showed no pathologic changes. The lens had not been dislocated. The depigmentation of the ciliary epithelium, which the author explained by maceration, had probably been caused by the trauma, together with the tear of the ciliary muscle and the rupture of the artery. The edema and hyperemia of the ciliary body and the accumulation of fluid in the suprachoroidea may be accepted. As to the accumulation of pigmented cells in the lymphatics around the blood vessels and the nerves in the sclera, one is inclined to think that these were chromatophores, which are normally present in all the sheaths and which have a lighter color than the pigment cells from the choroidal epithelium. It seems unlikely that the author has seen the same pigment granules that I have seen in the same place, as he clearly mentioned cells, not granules.

CASE 2 (Morax, 1921, eighteenth case).—An eye of a patient aged 65 was removed four weeks after an accident. The lens was subluxated anteriorly, with rupture of the zonule below. An albuminous, hemorrhagic exudate was seen in the anterior chamber. The iris was unrecognizable; only a few tracts and masses of pigment remained below, and rests of the pigment layer were adherent to the cornea above. At the periphery the iris lay against the cornea. The insertion of the ciliary processes seemed to be displaced backward. There was hemorrhagic infiltration of the lower half of the choroid. The outer layers of the retina were pathologically changed; the pigment epithelium had disappeared; the rods and cones were unrecognizable, and hemorrhages were seen in the layer of rods and cones and in the inner layers of the choroid. The inner granular layer and the layer of nerve fibers were in comparatively good condition and showed no hemorrhages. There were no signs of inflammation.

Although this eye showed a slight dislocation of the lens, Morax considered that this could not have been the cause of glaucoma. Morax also described a case of traumatic glaucoma in which the lens had been expelled through a rupture of the sclera. Obviously, the lens could not have caused glaucoma in that case, and no signs of inflammation could be observed.

CASE 3 (Lamb, 1927).—An eye of a patient aged 9 years was removed eight weeks after an accident.

The sclera was thin at the equator. The anterior chamber was full of blood, and the trabeculae of the filtration network were filled with red blood corpuscles. The iris was atrophic and about half the normal thickness. There was moderate proliferation of the chromatophores. The blood vessels were reduced to thin tubes, the majority even to capillaries. The number of muscle fibers of the sphincter muscle was diminished. The amount of albumin in the fluid in the stroma was increased. The posterior epithelium was thickened in some places, and there was loss of pigment in other places. In other places the epithelium was thin and interrupted. Here and there posterior synechiae were seen. The base of the iris had been torn through the pectinate ligament; the cleft that had thus been formed continued backward into the ciliary body just anterior to the ciliary muscle; much

blood was present in this gap, as well as in the surrounding tissue. The muscle of the ciliary body was thin; in the vascular layer many newly formed capillaries were full of polymorphonuclear leukocytes; in addition, a considerable number of plasma cells and proliferation of the chromatophores were seen here. The lens showed loss of anterior epithelium in the pupillary area and liquefaction of the fibers just behind this area. Outside the pupillary area a layer of fibers of the superficial part of the cortex containing vacuoles was seen; it ended behind the equator. The choroid was atrophic in its anterior and middle parts; many newly formed capillaries and proliferation of chromatophores were present. The retina showed loss of pigment of the pigment epithelium, cystic degeneration in its anterior half and marked proliferation of neuroglia cells in the posterior part. There were no pathologic changes of the optic nerve.

Lamb noted pathologic changes that corresponded in several respects to the changes that I observed (which will be described later), e. g., the changes in the iris, the rupture of the base of the iris into the ciliary body and also, perhaps, the changes in the choroid at the equator.

CASE 4 (Priestley Smith, 1881).—An eye of a patient aged 57 years was removed fifteen weeks after an accident. No blood was present in the eye. At the periphery the iris lay against the posterior surface of the cornea; there was coredialysis of 6 mm., and the iris had been torn completely from its base. The lens did not touch the ciliary processes. The ciliary muscle was perhaps somewhat atrophic. The optic nerve showed commencing excavation.

CASE 5 (Priestley Smith, 1883).—An eye of a patient aged 59 years was removed fifteen weeks after an accident.

In this case iridectomy was performed, after which the eye had to be removed immediately because of the enormous loss of vitreous. Large, darkly pigmented cells were scattered throughout the iris and its base. The angle of the anterior chamber was closed over a considerable distance. Schlemm's canal was partially filled with pigment; in some of the sections it was filled with erythrocytes. The lens showed rupture of the zonule medially and superiorly. The lens touched the ciliary processes. The relative positions of the iris, the ciliary processes and the lens were unreliable because of the operation, the loss of vitreous and the large hemorrhage. A large hemorrhage was seen outside the choroid and against the sclera. The optic nerve showed a deep but incomplete cup. The iris and the ciliary muscle were perhaps changed, but the difference between them and the normal structure was not obvious.

It is interesting to read that Priestley Smith observed that the iris and the ciliary muscle were normal or, at any rate, saw no obvious changes. The pigment granules in Schlemm's canal are best explained by a rupture of this canal.

CASE 6 (Leslie Buchanan, 1907).—An eye of a patient aged 59 years was removed six months after an accident.

Only the anterior half of the globe was examined. Rupture of the pectinate ligament and rupture of the inner layers of the sclera on the medial side were seen. Probably this represented a typical partial rupture of the sclera, originating in Schlemm's canal. In addition, ectasia was present at the equator, but its structure was not described in detail.

The most important of all these reports is Garnier's description. This author stated that the iris, as well as the angle of the anterior chamber, was normal, while the observations in his case bear a close resemblance to the microscopic observations in my cases.

REPORT OF TWO ADDITIONAL CASES

In two cases observed by me the globe was cut into serial sections. Every tenth section was then stained with iron hematoxylin or according to Van Gieson's method. Some of the intermediate sections were prepared later; whenever it was necessary, as in case of vessels, a complete series was examined. Special staining methods were employed to demonstrate the myelin sheaths of the nerves and to demonstrate the iron in the hematogenous pigment.

CASE 1.—*Clinical History.*—A. J. K., a shop assistant aged 17, stumbled and struck his left eye against the corner of a wooden box. He was examined the same day (Feb. 18, 1926). The right eye was normal and emmetropic, with visual acuity of 1.

The left eye showed a hematoma and superficial abrasions of the upper lid. There was extensive erosion of the corneal epithelium and of the bulbar conjunctiva. The anterior chamber was deep. The pupil did not react to light, and red light was reflected from the retina through the pupil. The tension was normal.

On the third day the patient had an intra-ocular hemorrhage and was admitted to the clinic.

On March 13 the tension was doubtful. On March 20 it was + 1, and the corneal epithelium was vesiculated. On March 29 the tension was + 2. On April 1 the globe was removed, as the sclera began to bulge superiorly.

Macroscopic Examination.—Even macroscopically the sclera was seen to be thin superiorly (fig. 1).

Microscopic Examination.—Cornea: The epithelium was irregularly thick. Its surface was rough; the nuclei of the cells were stained irregularly, and the cells were swollen. Here and there the epithelium had been lifted from Bowman's membrane to form vesicles of varying size.

Sclera (fig. 1): The sclera showed two ruptures of its inner layers—a typical rupture originating in Schlemm's canal superiorly and medially and an atypical rupture near the equator. These have been fully described elsewhere (Tillema, 1936).

Vessels in the Sclera: In the region of the incomplete rupture of the sclera Schlemm's canal had been destroyed. The veins of Schlemm's plexus were wide. They were surrounded here and there by small round cells and, especially below, by pigment granules containing iron.

Superiorly the wall of a ciliary artery had been disorganized (fig. 2). The lumen of the vessel was filled with a fine granular material, and its endothelium seemed swollen. The surrounding sclera showed the changes that have already been described elsewhere and which represent ruptures of the scleral fibers. Somewhat farther along blood pigment was seen in the wall of the vessel and in the sclera around the artery. The anterior branch running in the ciliary body was seen to be damaged, while the posterior branch had been obliterated and organized.

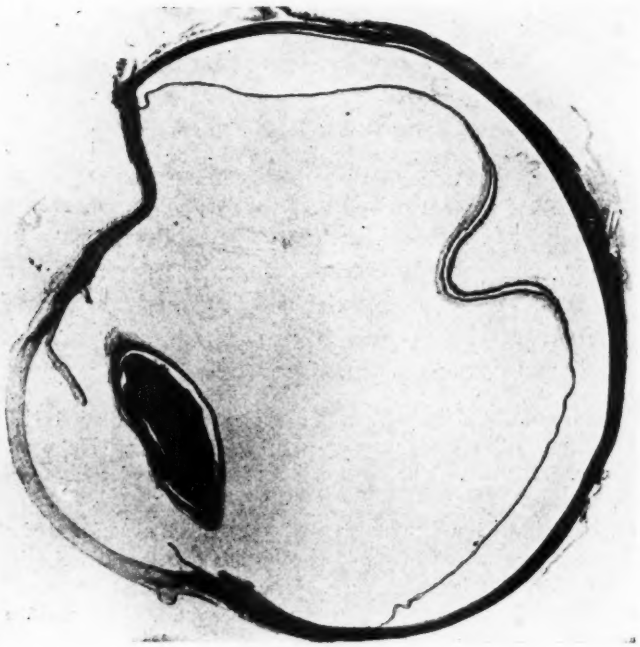

Fig. 1 (case 1).—General view of the injured eye. Note the paleness of the sclera above (incomplete, atypical rupture) and its thinness near the angle of the anterior chamber (incomplete, typical rupture). Also note the asymmetry of the anterior chamber.

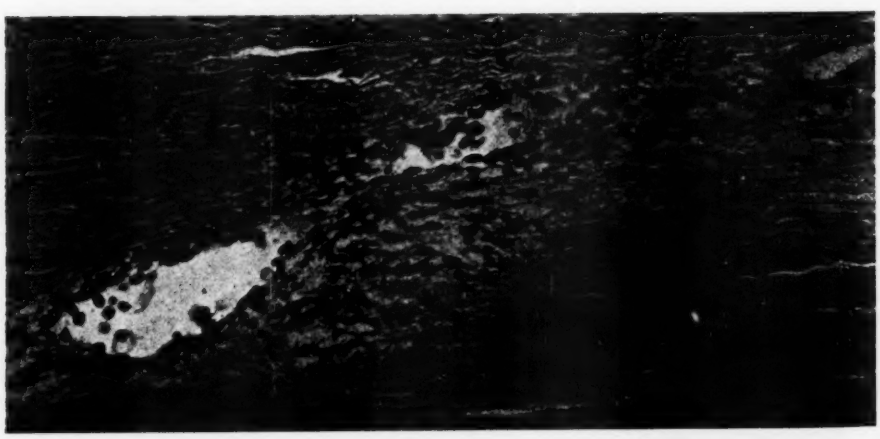

Fig. 2.—A ciliary artery. The wall of the vessel is partly disorganized. The surrounding sclera is very irregular in structure and contains scattered granules of both uveal and hematogenous pigment. Below, the ciliary body is just visible.

Another artery, on the inner nasal side, showed identical changes plainly. While it was intact in the outer layers of the sclera, its front wall in the inner layers of the sclera consisted of endothelium only (fig. 3), and a little farther it even showed a defect.

Analogous changes were seen in many other places in the anterior ciliary vessels, both in arteries and in veins.

The short posterior ciliary arteries showed no changes. One of the long posterior ciliary arteries, on the other hand, was seen to be interrupted in the inner

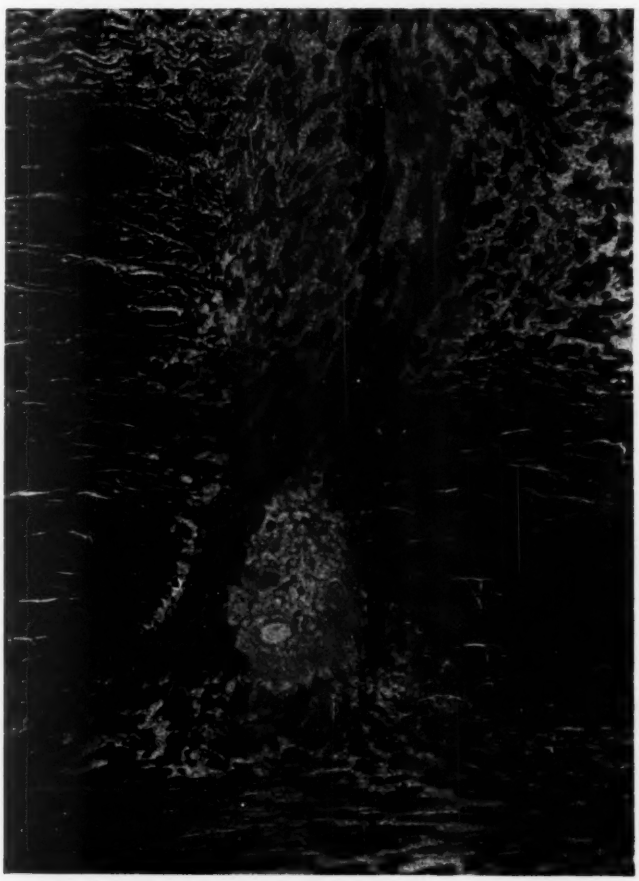

Fig. 3.—An anterior ciliary artery, showing on the right a strikingly thin wall. The wall shows a defect on the right and below. The ciliary body is seen below.

layers of the sclera. While it was well preserved in the outer layers of the sclera, with its accompanying nerve, in the inner layers of the sclera only an irregular mass of pigment granules was seen close to the nerve (figs. 4 and 5), but no vessel.

Five vortex veins were noted in the sections. The inferior nasal vein showed endothelial defects, which were covered with clustered white blood cells containing round and segmented nuclei. The surrounding fibers of the sclera ran a slightly irregular course. A few small round cells were scattered between them. Close to

the choroid the wall of the vessel was infiltrated by small round cells. Between the cells pigment containing iron was scattered. Slightly deeper in the choroid the wall of the vessel and the surrounding sclera contained a striking number of small round cells. Between and inside the endothelial cells of both the superior and the inferior temporal veins pigment granules were seen in a few places (fig. 6). The

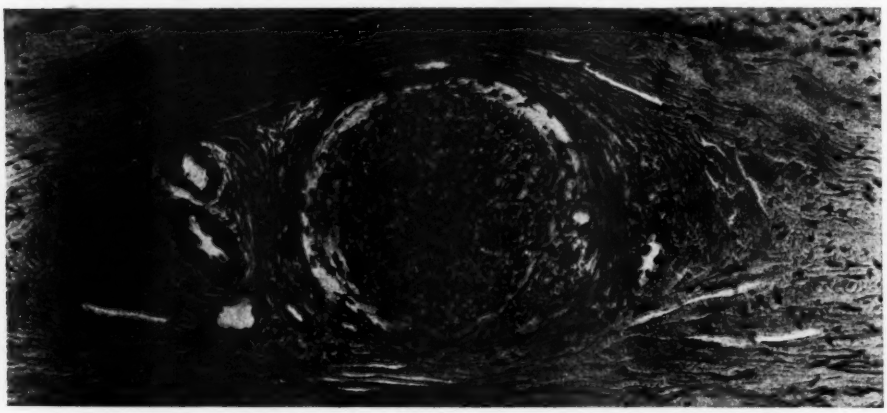

Fig. 4.—Posterior ciliary vessels and nerve in the outer layers of the sclera. Note the normal structure of all the tissues and compare with figure 5.

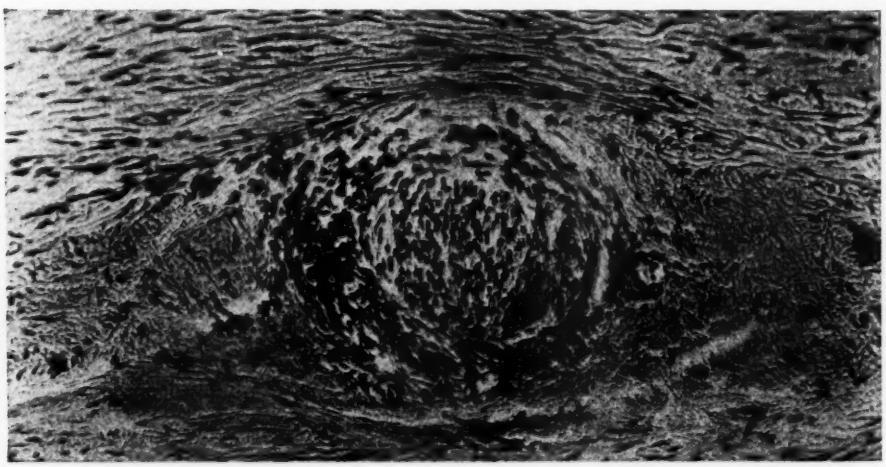

Fig. 5.—Same vessels and nerve as in figure 4. The vessels have been replaced by pigment (this is a section through the inner layers of the sclera, about 0.8 mm. from the section shown in figure 4).

other three veins were normal. However, pigment granules were observed in the perivascular channels of all five veins.

Ciliary Nerves: The ciliary nerves in general showed good structure and good staining properties. In a few instances, however, isolated groups of myelin sheaths were found to have degenerated, while the rest of the bundle was well preserved.

Anterior Chamber: The anterior chamber was remarkably deep. As the distance from the end of Descemet's membrane to the anterior ciliary processes was one and one-half times as large superiorly as inferiorly, the sclera must have been stretched in the same region (incomplete rupture of the sclera; see this section). The cornea, therefore, had been displaced forward. This explains the unusual depth of the anterior chamber. It also explains why the anterior chamber was deeper superiorly than it was inferiorly (fig. 1).

The iris had closed the angle of the anterior chamber everywhere. It was adherent to the cornea. The new-formed angle contained a small amount of granular material inferiorly. Superiorly the pectinate ligament had been greatly stretched, and some of the trabeculae were interrupted. Between the trabeculae many pigment granules were seen, and small round cells were noted. This pigment was largely hematogenous.

Iris: The anterior surface was torn superiorly. The stroma had lost a great deal of its normal structure in all its parts. It had taken the stains poorly, and

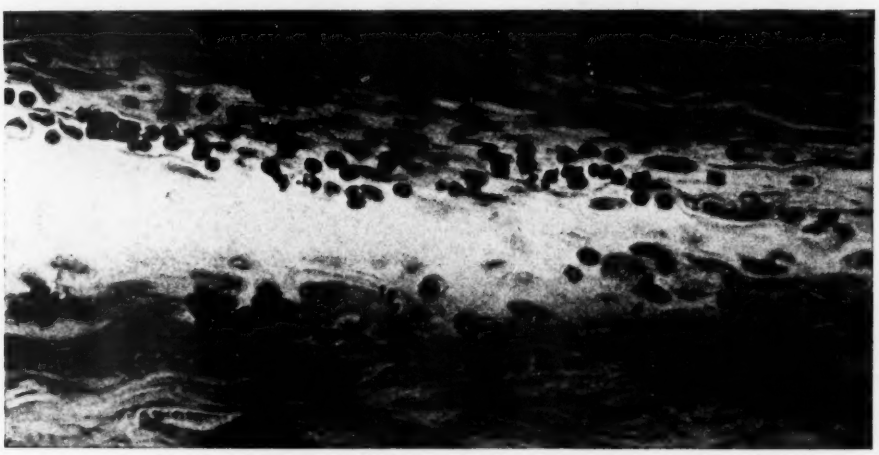

Fig. 6.—A vortex vein, showing pigment granules inside the endothelial cells.

it contained a great number of scattered lumps of pigment which were mostly hematogenous. The nuclei of the cells were pale, shrunken or disintegrated. Especially below, the stroma was almost structureless and had been replaced by a material that stained almost uniformly with eosin (necrosis of the iris; fig. 7). Below, near the ciliary body, the pigment in the posterior portion was scattered, mainly into the posterior chamber, but also into the stroma of the iris. A few giant cells with multiple nuclei were observed here.

The base of the iris was thin everywhere. It consisted almost exclusively of pigment epithelium. Inferiorly the iris had been split off the ciliary muscle. The new-formed angle of the anterior chamber continued backward into the ciliary body and contained a large amount of hematogenous pigment.

Ciliary Body: At the ora serrata the transition of the pars plana into the choroid had been destroyed. The changes seen in this region corresponded to those which will be described in the section on the choroid.

Inferiorly the ciliary processes had been partly destroyed. A large amount of pigment was lying scattered in the tissue of the ciliary body, partly in big lumps

and partly in fine granules. These fine granules were in part hematogenous. Here and there the pigment was arranged in layers, probably filling tissue spaces (necrosis of the ciliary body). Here and there, between the ciliary processes, small bundles of elongated cells were seen. It was difficult to decide whether these were fibroblasts or cells stretched by traction and belonging to the ciliary epithelium.

The pars plana of the ciliary body was separated from the sclera by a space containing fluid. Inferiorly this cleft was much more extensive than above, and it continued in the suprachoroidea as far back as the equator. In a lateral direction it was observed in nearly all the sections.

Choroid: Nasally and above, the choroid had been almost completely depigmented. In this region it was composed of loose cells and lumps of pigment

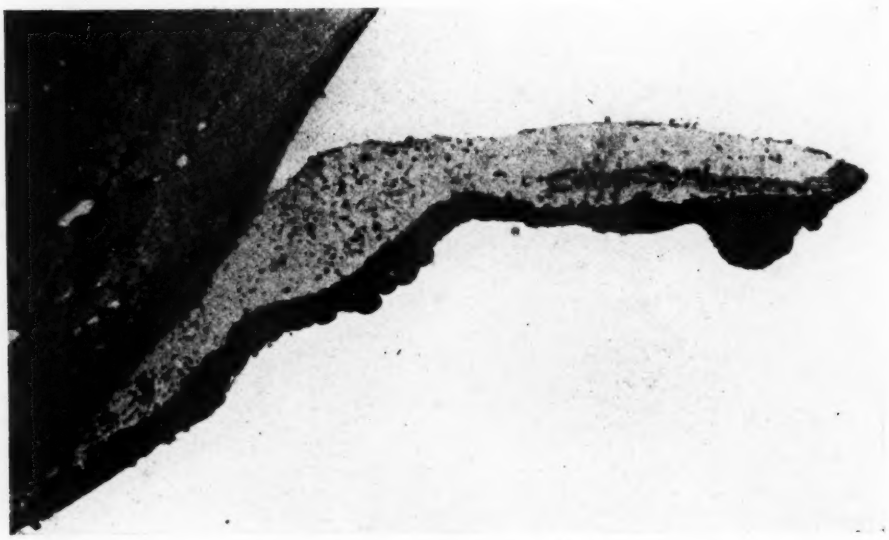

Fig. 7.—The iris, showing necrosis, with loss of structure and pigment granules scattered throughout the stroma. The angle of the anterior chamber is closed.

interspersed with spindle cells and a few small round cells (rupture of the choroid; fig. 8).

It has been mentioned already that the fluid-containing space between the ciliary body and the sclera continued backward outside the choroid (fig. 8) as far as the equator. Posteriorly also, however, the suprachoroid contained fluid, but only a small amount. Close to the optic papilla, especially laterally, the capillary layer of the choroid and the layer of middle veins had been replaced by closely packed spindle cells the nuclei of which stained well. These cells had broken through Bruch's membrane, and they were surrounded in the choroid by groups of small round cells (scar of a circumpapillary rupture of the choroid).

Lens and Zonule: The capsule of the lens was intact but slightly wrinkled. The fibers of the lens at the equator were well preserved and showed well staining nuclei. Centrally, both anteriorly and posteriorly the fibers had broken down to form a fine granular material interspersed with globules.

The zonular fibers were broken here and there and were markedly wrinkled.

Retina and Optic Nerve: Close to the ora serrata the retina became thinner, and the granular layers ran together. At the ora serrata, nasally and above, the retina was hardly recognizable (rupture of the retina at the ora serrata; fig. 8).

In many places close to the pigment epithelium small structureless globules were seen. As these were better seen in the second case, they will be described in the report of that case.

In the macula lutea the granular layers were completely broken, while pigment and pigment cells lay scattered through the outer retinal layers (hole at the macula).

The optic nerve had a deep funnel-shaped cup. This cup was narrow and extended through the lamina cribrosa into the optic nerve. It was impossible to determine how far it extended into the optic nerve, as the latter had been severed close to the globe. The columns of the fibers of the optic nerve anterior to the

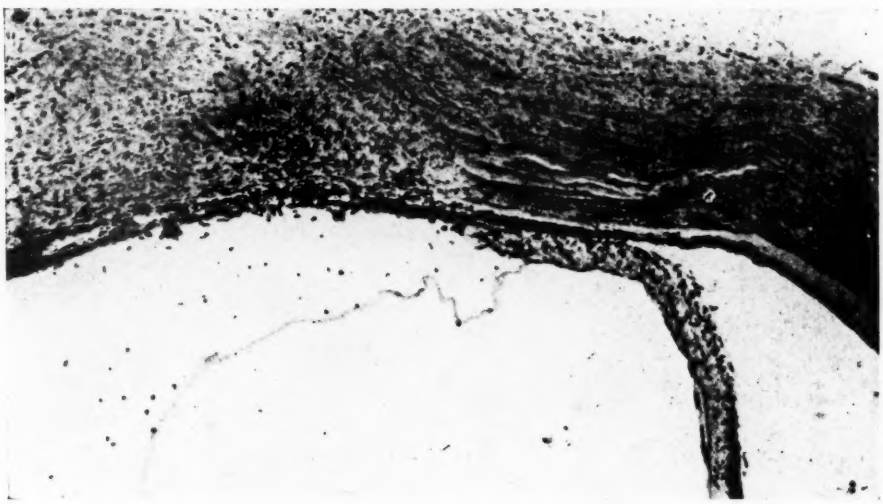

Fig. 8.—The sclera, choroid and retina. The choroid and retina are torn in the same region where the sclera exhibits marked changes in structure and staining. Note the fluid in the epichoroid.

lamina cribrosa appeared to be dislocated backward (rupture of the optic papilla associated with rupture of the lamina cribrosa, Tillema, 1936).

Vitreous Body: The vitreous was closely filled with erythrocytes the contours of which were just visible under high magnification. In addition fine granules staining with eosin and lying close together were observed everywhere. Between these granules many cells were seen to be in varying stages of degeneration. The majority possessed an oval, eccentric nucleus and a finely granular protoplasm. Some had vacuoles, others contained dark brown pigment granules. In a few places, near the ora serrata, the pigment could be demonstrated to contain iron.

CASE 2.—*Clinical History.*—T. de L., a manual laborer aged 30, received a blow on the left eye with a piece of wood. He was examined the same day (April 13, 1931).

The right eye was normal and emmetropic, with visual acuity of 1.

The left eye showed a fairly large hematoma of both eyelids and the surrounding tissues. The lids were torn in several places. The bulb was hardly seen through the swollen lids and the conjunctival edema. The cornea was hazy and the pupil wide; the iris was perhaps torn. The patient bled from the nose, complained of dizziness and vomited but refused to be admitted to the clinic.

On April 21 he was admitted to the neurologic clinic because of signs of commotio cerebri. Nothing serious was found, however, and the next day he was transferred to the ophthalmologic clinic because of the raised tension of the eye.

The cornea was then steamy and the pupil wide and irregular. Behind the pupil the movable, cataractous lens could be seen. The tension was from + 2 to + 3.

Fig. 9 (case 2).—General view of the eye. The sclera is thinner nasally (left) than temporally. The ciliary body is very thin, and there is a glaucomatous cup. The anterior displacement of the lens is an artefact.

On May 9 the patient left the clinic of his own accord, although he was urged to stay. Some time later he returned. The tension was then + 3, and the globe had to be removed (June 18).

Microscopic Examination (fig. 9).—Cornea: The epithelial surface was irregular. Here and there the superficial cells appeared to be missing, while in other places they were swollen and vacuolated. The vacuoles had fused and had formed vesicles of varying size. The largest were as big, approximately, as a few cells put together.

Sclera: The sclera had been incompletely ruptured anterior to the equator. This rupture has been described in detail elsewhere (Tillema, 1936).

Vessels in the Sclera: Schlemm's canal had been obliterated in one place medially. It had been replaced by spindle-shaped cells and cells with a large, pale, oval nucleus and by a little pigment.

The anterior ciliary vessels showed pathologic changes in many places. This was well illustrated by an artery running from the internal rectus muscle through the sclera into the ciliary body. In the belly of the muscle the vessel was unimpaired. Near the insertion it was surrounded by closely packed round cells.

Fig. 10.—An anterior ciliary artery penetrating the sclera. The lumen is destroyed, and pigment (both uveal and hematogenous) is scattered in the surrounding tissue. Note the irregular structure of the sclera surrounding the vessel near the ciliary body (below). Above, the artery branching off to the right ends abruptly.

Slightly more anteriorly spindle cells and one or two pigment granules were also observed. The endothelium was swollen, and the wall of the vessel was damaged. Nearer to the sclera these changes became more marked, while at the point of entry (fig. 10) into the sclera the lumen of the vessel was missing. Connective tissue, spindle cells, cells with a large, pale nucleus and small round cells occupied the site of the vessel. In the inner layers of the sclera the same picture was seen, but here a great deal of pigment was scattered about. This pigment was to a

large extent hematogenous. The surrounding sclera had taken the stains poorly and contained various cells and scattered pigment. An anterior branch of this artery had also been obliterated (fig. 10).

One other spot, in the lateral portion, deserves description. Just in front of the anterior ciliary processes the ciliary epithelium had been depigmented and almost interrupted. Large cells containing pigment were lying loose in the posterior chamber and in the ciliary body. If one traced this spot in the serial sections one observed pathologic changes in the inner scleral lamellae similar to those described under partial rupture of the sclera elsewhere (Tillema, 1936). Still farther the remains of a blood vessel were seen, and in tracing this one noted that it was intact in the outer layers of the sclera (rupture of the ciliary body and the inner layers of sclera at the point of entry of a blood vessel).

In the long posterior arteries also pathologic changes were noted. The nasal artery suddenly showed remarkable thinness and poor structure of its wall in the inner layers of the sclera (fig. 11). The temporal artery had no lumen at its point of entry into the choroid, while its endothelium was missing. Its wall had taken the stains badly here and had an irregular structure (fig. 12).

Fig. 11.—A long posterior ciliary artery (nasal artery), showing an exceedingly thin, almost structureless wall in the inner layers of the sclera.

Four vorticose veins were present. Their perivascular channels were filled with hematogenous pigment. In the inner layers of the sclera, however, this pigment was mixed with pigment derived from the choroid. In one vein the pigment was seen lying in the wall of the vessel against the erythrocytes filling the lumen of the vessel (fig. 13). In another vein pigment was seen lying inside the lumen (fig. 14), while a little farther erythrocytes were seen to fill the spaces surrounding the vein (fig. 15). While the first three veins showed small endothelial defects covered with groups of white blood cells, the fourth vein showed a complete defect of its wall close to the choroid. The surrounding sclera was stained irregularly and contained spindle cells and granules of blood pigment.

The pigment which was seen in the perivascular channels of all these veins could be traced outward into the episclera and from there backward toward the posterior pole of the eye (fig. 16).

Ciliary Nerves: There was a notable contrast between the good structure of most nerves and the partial degeneration of others. The isolated degeneration of one group of myelin sheaths, with good preservation of the rest of the bundle, is demonstrated in figure 17.

Anterior Chamber: The anterior chamber had been completely deformed through the anterior displacement of the iris and lens. The iris, at its periphery, was adherent to the cornea and had closed the angle of the anterior chamber.

Fig. 12.—A long posterior artery (temporal artery). The lumen is closed; the endothelium is missing, and the wall is irregular in structure (section through the inner layers of the sclera).

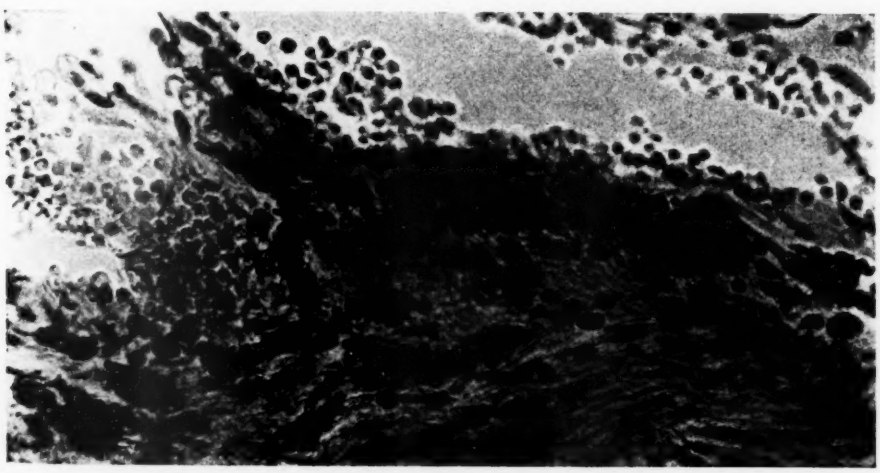

Fig. 13.—A vortex vein, showing erythrocytes inside the lumen and pigment granules in the wall touching each other.

Nasally the vitreous body had entered the anterior chamber and was seen in front of the lens. Continuing on its way in a temporal direction and passing between iris and lens, it had entered the posterior chamber temporarily. The anterior vitreous membrane in this region was complete.

In addition to the vitreous the anterior chamber contained only a few pigment-laden cells.

Iris (fig. 18): The iris was adherent to the cornea peripherally and had closed the angle of the anterior chamber. Its tissue had taken the stains poorly and showed little structure. The nuclei of the cells had a varying depth of color

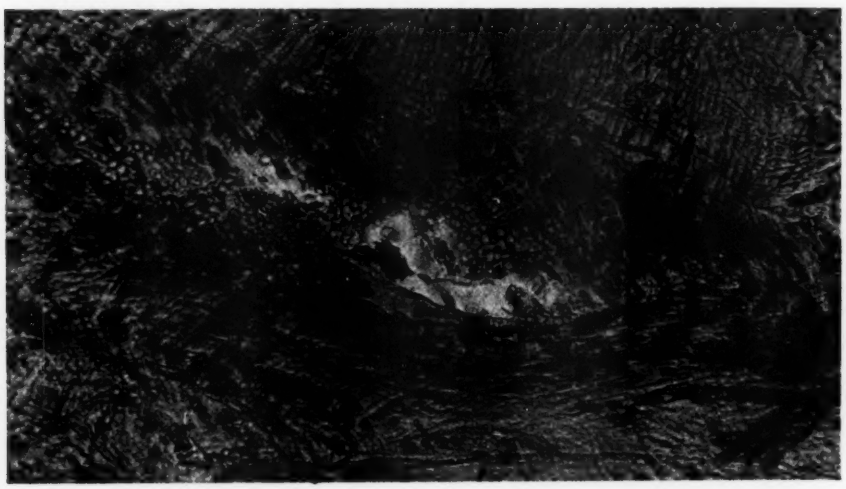

Fig. 14.—A vortex vein, showing pigment granules inside the lumen.

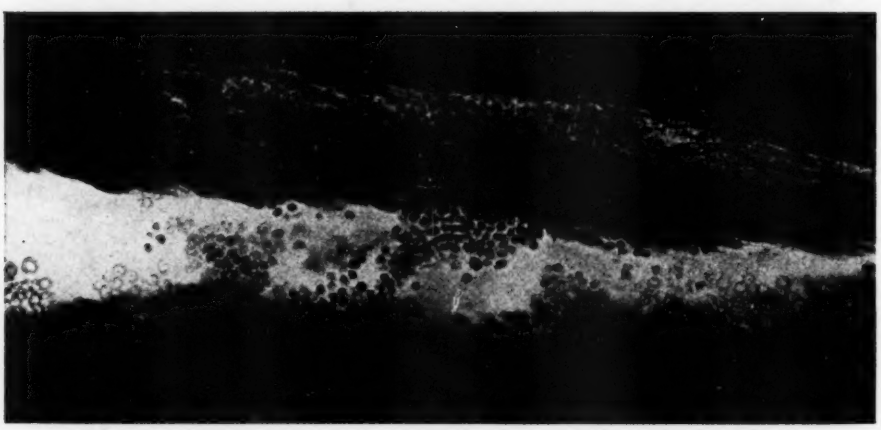

Fig. 15.—The same vortex vein as in figure 14. The lumen of the vein is seen below. Above are seen perivascular channels filled with erythrocytes.

or had shrunk; the walls of the vessels were stained homogeneously with eosin, while the endothelium was swollen or missing. Laterally a large piece of the uveal portion of the iris was missing. In other places medially the whole sphincter portion was missing, while the pigment layer had curled up. In other places tears of the dilator and sphincter muscles and of the stroma were seen.

Ciliary Body: Nasally the epithelial pigment had been scattered into the posterior chamber and into the surrounding tissue of the ciliary body. It was seen as large, round lumps (loose epithelial cells?) in the posterior chamber; in the epithelium and in the surrounding tissue, on the other hand, it appeared as fine, diffusely spread granules. In sections stained for iron no iron was observed here, although it occurred in many places between the ciliary body and the sclera.

Temporally, in a circumscribed area, the anterior ciliary processes contained dense connective tissue with few nuclei and a small amount of scattered pigment.

Fig. 16.—A vortex vein (stained for iron). Above, the vein is seen close to its point of exit from the sclera. Note the closely packed hematogenous pigment around the vein. It may be traced toward the posterior pole (left) of the globe.

Fig. 17.—A ciliary nerve. On the right are well stained myelin sheaths. On the left is a larger bundle in which the myelin sheaths are partly missing.

Just anterior to these processes the epithelium had been destroyed. The ciliary body was very thin here, and bending outward it lined a scleral pit. Traces of iron were noted in this region. Under the section on the ciliary vessels I have described the presence of a damaged vessel in this part of the sclera (rupture of the ciliary body at the point of entry of a ciliary vessel).

The ciliary muscle as a whole was very flat and appeared to contain much connective tissue. In some places pigment granules were grouped around a vesel, generally in the neighborhood of tissue changes.

Choroid: Nasally and above, the choroid and the retina had been destroyed. They had been replaced by connective tissue showing many elongated dark nuclei. This tissue gradually merged into the sclera, which was pale in this region and contained many nuclei (rupture of the choroid).

Fig. 18.—The iris, showing necrosis, a tear of the anterior lamella and a thin base adherent to the cornea.

Fig. 19.—The capsule of the lens and the anterior part of the vitreous membrane, both covered with cells derived from the epithelium of the lens.

From the ora serrata backward to a distance far behind the equator the pigment of the epithelium and the chromatophores had been scattered. This was more marked nasally. In a few places the choroid had been completely depig-

mented; it was thin, and consisted of a few elongated cells with spindle-shaped nuclei.

Temporally and behind, smaller ruptures of the choroid were observed. They were represented by groups of closely packed spindle cells which had broken through Bruch's membrane and through the pigment epithelium. Between these cells iron was seen. The outer layers of the choroid had been fairly well preserved. The retina, which had been artificially detached, was adherent to the choroid here.

The outer layers of the choroid and the epichoroid were closely packed with pigment granules. This pigment contained a great deal of iron.

Lens and Zonule: The lens had shrunk. It had been displaced anteriorly (post mortem), and its capsule had been torn. The epithelium had grown around the rent along the hyloid membrane and along the zonule (fig. 19). The normal structure of the lens had been largely destroyed: its substance was granular, and

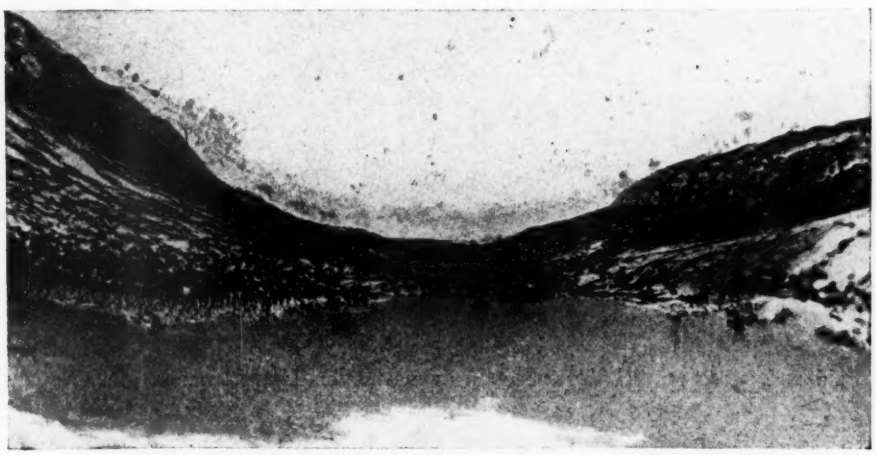

Fig. 20.—The macula lutea. The cones are missing in the fovea. Note the pigment granules in the fovea and the subretinal fluid.

the epithelium was missing in many places. The meridional zonular fibers had been torn nasally, while the radial fibers were missing. Only remnants of the latter were observed temporally.

Retina: The retina was torn in the same place as the choroid. In the macula lutea the cones were missing. Pigment cells and loose pigment were scattered throughout the remaining layers (fig. 20).

The neuro-epithelium was separated from the pigment epithelium by fluid (fig. 21). The neuro-epithelium at the equator had degenerated into small globules of varying size and shape (fig. 21). The majority were round and stained with eosin and with fuchsin. Interspersed between these globules lay large cells with an oval or kidney-shaped pale nucleus. They contained pigment, partly hematogenous but partly also resembling the pigment of the pigment epithelium. Both the globules and the large cells were observed close to the pigment epithelium. The pigment epithelium was swollen. A few cells had even assumed a semi-globular shape (fig. 22) or had formed into a large structureless mass resembling the small loose globules. Just as in the choroid, the changes in the retina were more marked nasally.

Optic Nerve: The optic nerve has been fully described elsewhere (Tillema, 1936).

The optic papilla and the lamina cribrosa had been incompletely ruptured in a backward direction. In this region the central vein had also been torn, and the vitreous protruded into it. In addition, there was a glaucomatous cup.

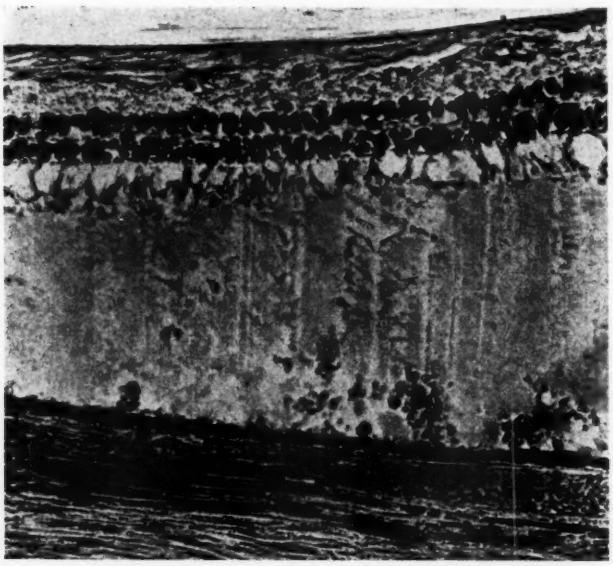

Fig. 21.—The retina. The rods and cones are missing. Note the subretinal fluid and, close to the pigment epithelium, the small structureless globules.

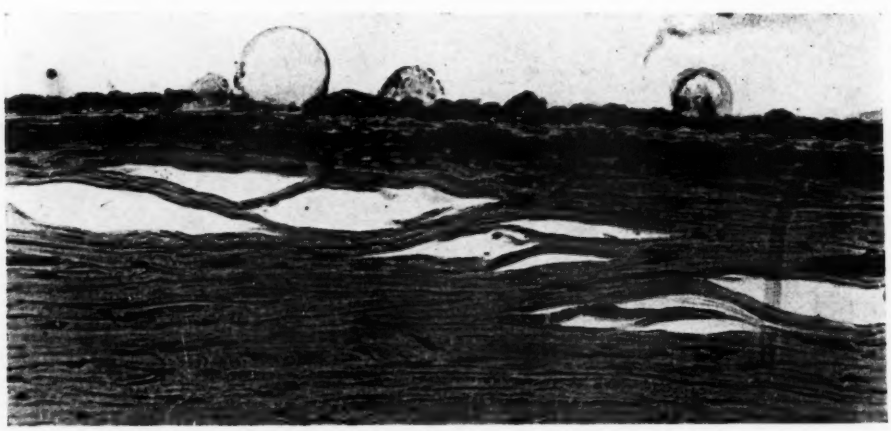

Fig. 22.—Pigment epithelium, showing "inflated" cells.

Vitreous Body: The anterior hyaloid membrane had been torn medially. The vitreous at this point had entered the anterior chamber.

The vitreous contained many scattered cells, mostly with a nucleus varying in shape from oval to segmented. The cells carried many pigment granules that con-

tained no iron. The space between the cells was taken up by a fine granular pink material with small round cells here and there. Many cells showed signs of degeneration to a greater or lesser degree.

Near the equator of the lens and also anterior to the lens in the anterior layers of the vitreous a circumscribed group of red and white blood cells was seen.

COMMENT

Although the two globes just described differed in small details, their resemblance is obvious. No signs of a perforating wound or of infection were noted. The pathologic changes may conveniently be divided into two groups: a large group of destructive changes and a smaller group of regenerative changes. It is obvious that the regenerative changes could not but have been preceded by the destructive changes and that the latter might have been caused partly by the trauma, but that they might also have arisen through unfavorable physical or chemical conditions. As in the majority of cases the changes occurred immediately after contusion (see "Clinical Part"), those changes that were directly caused by the trauma were most likely to give the key to the problem of traumatic glaucoma. For this reason this group of changes will be separated from the others.

All formation of new vessels, all formation of scar tissue and all transport of débris must be regarded as regenerative changes. Formation of new vessels was seen only in the cornea in the first case. Formation of scar tissue was seen in the choroid and sclera. Transport of débris, however, was seen in many places, especially in the vitreous and around the vessels in the sclera. The numerous loose pigment granules were partly hematogenous and partly derived from the uvea. Although both kinds usually occurred together, it must be noted that the pigment around the vorticose veins was almost exclusively hematogenous. It was interesting to see the growth of lenticular epithelium (my second case) along the torn capsule and the anterior part of the hyaloid membrane. This illustrates that the lens too is a living tissue.

The remaining changes were destructive. As some of them may be observed in eyes that have never suffered a trauma, it seems unlikely that they should constitute a primary factor in the origin of traumatic glaucoma. For this reason these also must be separated and laid aside.

The changes which have been observed in the corneal epithelium and stroma are familiar. They have often been described as regards both their clinical aspects and their microscopic aspects in cases of glaucoma. The changes in the sclera have been fully discussed in a separate article and constitute indirect, incomplete ruptures. Adherence of the peripheral part of the iris anteriorly, thinness of its basis and infiltration by pigment of the trabeculae of the pectinate ligament have often been observed preceding intercalary staphyloma in cases

of primary glaucoma. Moreover, according to the clinical history in my cases, the adherence of the peripheral part of the iris did not exist at first. This, then, must be regarded as a secondary change, a view supported by Garnier's observation that in his case the angle of the anterior chamber was normal.

Necrosis of the iris is seen in association with many different conditions. I know from personal observation that it may occur in advanced stages of glioma of the retina with secondary glaucoma, in cases of choroidal sarcoma and in cases of leprosy. It has been described (A. Fuchs) as occurring in cases of ulcus serpens corneae, in cases of traumatic ring abscess of the cornea associated with endophthalmos, in cases of necrotic sarcoma of the choroid and in cases of glaucoma. Necrosis of the iris, therefore, may be a secondary change, but it has been described after contusion (A. Fuchs). Moreover, the hematogenous pigment scattered irregularly throughout the stroma of the iris suggests a traumatic lesion. Another reason for thinking this is the fact that the ciliary body exhibited similar changes in a circumscribed area (my first case), while in glaucoma the changes would be more general. If the pigment granules in the iris were grouped around the vessels one might be tempted to think that the vessels had formed drainage channels for the pigment, such as Gerbrandy hypothesized from his investigation or such as Rochat observed in his experiments with hemoglobin. There is no such grouping, however, and therefore the pigment granules must be remnants of hemorrhage. Finally, I have observed necrosis of the iris in a case of contusion caused by an exploding cartridge. One may conclude, therefore, that at least part of the necrosis of the iris had been caused by contusion.

In my first case both the anterior chamber and the posterior chamber had been deformed by displacement of the iris and lens. The wrinkling of the capsule of the lens suggests a slackening of the zonule in vivo, a likely view if one remembers that the zonular fibers are partly torn. Probably, therefore, the lens was slightly dislocated in vivo. In my second case the anterior forward displacement of the lens must have been an artefact.

The circumscribed necrosis of the ciliary body (my first case) has already been mentioned. The same arguments brought forward for the iris apply here. The thinness of the ciliary body and its elongation in a backward direction may have been caused partly by distention of the sclera but partly also by atrophy such as is seen in glaucoma.

Detachment of the choroid and of the ciliary body is also observed after intra-ocular operations and generally in hypotony of the globe. It is remarkable, therefore, that it occurred in both my cases and in Garnier's case of traumatic glaucoma. It may be explained by disturbance of the intra-ocular circulation.

The cupping of the optic papilla (my second case) is similar to the cupping in glaucoma, but in addition both my patients showed a tear of the lamina cribrosa and the optic nerve. The latter must have been caused by contusion (Tillema). Structureless globules between the pigment epithelium of the retina and swelling of the epithelial cells such as I have seen have been described by Koyanagi in cases of nephritic retinitis and eclampsia. From the observation of intermediate stages between the cells and the globules Koyanagi concluded that in his cases the globules had been actively secreted by the epithelium. As I did not observe such intermediate stages, the globules in my cases must be regarded as a form of degeneration based on circulatory disturbance in the choroid such as was described by Krückmann in 1899.

Thus one arrives at the last group of changes, i. e., those caused directly by contusion. In both cases this group consists of rupture of the inner layers of the sclera inward and upward, rupture of the lamina cribrosa, destruction of Schlemm's canal medially, lesions of the ciliary vessels and nerves, endothelial changes in the vortex veins, tears of the iris and (partly) its necrosis, circumscribed necrosis of the ciliary body and rupture of the retina and choroid. In addition, my second patient showed rupture of the anterior capsule of the lens and of the anterior hyaloid membrane, with prolapse of vitreous into the anterior chamber, and, finally, rupture of the central vein at the optic papilla. These differences between the two cases are important.

Although it is well known that both rupture of the capsule of the lens and thrombosis of the central vein may be followed by glaucoma, these must be exceptional causes of traumatic glaucoma, as neither rupture of the capsule of the lens nor rupture of the central vein was present in one of the cases collected from the literature and in one of my clinical cases (See "Clinical Part").

Some of the changes common to both cases also require further discussion. The lesions of the vessels in the sclera were so unusual that they could have been caused only by trauma. Nevertheless, changes in the vortex veins have also been observed in other forms of glaucoma. Hypertrophy of the endothelium obliterating the lumen of the vessels and obliteration of the perivascular channels through hypertrophy of tissue with few nuclei have been described, but these changes are not constantly present (Stirling, Köllner and Thomsen). According to Elschnig, they are more frequent and more marked in cases in which the condition is of long standing, and it has not been proved that these changes cause glaucoma. All these changes, however, are entirely different from what I observed. Partial closure of the veins without anatomic changes such as was described by Schieck (1934) in a case of malignant glaucoma is also different.

It is possible, however, that in traumatic glaucoma the veins may be closed by thrombi caused by a traumatic lesion. It has to be decided, therefore, whether the material observed in some of the veins represents thrombi.

It will be remembered that a lesion in the wall of a vessel must be accompanied by circulatory disturbance to cause formation of a thrombus (Dietrich, 1932). Possibly these two factors were present, but even so the absence of organization which is generally easily recognized after even a few days (Dietrich) renders a thrombus unlikely. Even if organization could be accepted in a few places this organization was so little marked that the thrombi must have been very young and could not have played a part in raising the intra-ocular tension. It is almost certain that they represented postmortem clots.

The granules of hematogenous pigment scattered around the anterior ciliary vessels and the vortex vessels must have been remnants of previous hemorrhage. They confirm the previously expressed opinion that the vascular lesions were traumatic. These granules were also observed in the iris and ciliary body, and they appeared to have been transported from the inner part of the eye along the epichoroidal and perivascular channels of the vorticose veins into the episclera. Thence they could be traced back toward the posterior pole of the globe.

Degeneration of the ciliary nerves also occurs in nontraumatic glaucoma. Isolated degeneration of a few bundles, however, in a nerve the other bundles of which have been well preserved is a striking phenomenon. There is no reason why this should occur in other forms of glaucoma, and it is suggestive of a traumatic lesion.

In summary, one may say that the following anatomic changes are probably directly related to the accident: partial rupture of the sclera, pectinate ligament, lamina cribrosa and optic nerve; tear and partial necrosis of the iris; necrosis of the ciliary body; rupture of the choroid and retina; rupture of ciliary and vortex vessels in the inner layers of the sclera (probably related to rupture of the sclera); subluxation of the lens (my first case), and isolated degeneration of bundles of the ciliary nerves.

The hemorrhage into the vitreous had been caused by vascular lesions and might have occurred immediately or later. The same applies, of course, to other hemorrhages.

Having analyzed the results of the anatomic study, one is tempted to propound a theory on the origin of traumatic glaucoma. The reader will have noticed, however, that even in the present discussion it has been necessary to refer to data that have issued from clinical study.

It is better, therefore, to await the result of clinical study and to discuss the anatomic data with the clinical data.

II. CLINICAL PART

REPORT OF FIVE CASES OF TRAUMATIC GLAUCOMA

After examining the microscopic sections one is, of course, interested to know whether similar changes can be seen in the living subject. I have been fortunate in having had the opportunity of examining a number of patients with traumatic glaucoma, although this condition is not often seen. The cases have been arranged according to the severity of the attack.

CASE 3.—T. J. S., a fitter aged 45 years, was hammering a brass tube when a piece of brass struck his eye with considerable force. He was examined the same day (March 18, 1933).

The right eye was normal, and visual acuity was 1. Refraction showed hypermetropia of 0.5 D.

The superior lid of the left eye was swollen, and the bulbar conjunctiva was torn medially and laterally. There was a deep wound of the cornea laterally and various erosions. Descemet's membrane was wrinkled. The iris was torn medially. The pupil was wide, and blood coagulum was seen adhering to the iris. The lens was clear on transillumination. No gross changes were seen in the fundus. Sideroscopic examination gave negative results.

On March 20 the lens was seen to be mobile; there was blood, but no exudate, in the anterior chamber.

On March 24 the lens turned diffusely turbid and began to move backward medially. The tension was slightly too high (+ ½).

On March 31 the tension was from + ½ to + 1.

Notwithstanding the administration of pilocarpine the tension rose higher. The patient was then admitted to the clinic, and the lens was extracted. Recovery was uneventful.

The vitreous cleared gradually. The patient was last seen on July 31, 1934. The cornea was then clear, and the anterior chamber deep. The pupil was wide, and it did not react to light or in convergence. A large artificial coloboma of the iris was present medially and superiorly. The stroma of the iris was very atrophic (both the anterior and the posterior lamella). The light reflected from the retina was seen through fissures in the stroma of the iris. The vitreous body was clear except for a few opacities. The fundus was well seen through the wideness of the pupil. The optic papilla was normal. The macula showed irregular pigmentation occupying an area slightly larger than the macula. The retina and choroid were normal except in the superior nasal quadrant, where the pigment of the ora serrata was scattered on a gray background showing a few white spots. A short distance behind the ora serrata also a small amount of pigmentation was seen. This pigmentation was coarse and was probably choroidal. The ciliary processes had been partly depigmented; they were seen as grayish-white masses alternating with darker masses. Part of the pars plana of the ciliary body was also seen.

The ocular tension was normal, as was the projection of light. Visual acuity was 1/300 and was but slightly improved by glasses. The field of vision revealed a large central scotoma.

In this case the ocular tension rose on the sixth day after contusion. The slight medial dislocation of the lens was probably only a coincidence. The rupture of the zonule, the prolapse of vitreous into the

anterior chamber, the medial tear of the iris, the hemorrhage into the vitreous and the pathologic changes of the retina and choroid indicate that this eye suffered a serious lesion. As many of the lesions were situated medially and superiorly, they must have been related. If one remembers that the majority of ruptures of the sclera are also situated superiorly and medially and if one recollects the description of the microscopic features in my first two cases, one is struck by their similarity. In every case pathologic changes were most marked superiorly and medially. In every case the changes in the retina and choroid consisted of loss of structure, pigmentation and slight formation of scar tissue. Changes were also seen in the ciliary body. One may be sure, therefore, that the clinical and the microscopic pictures correspond and that the clinical picture may be interpreted as representing a tear of both the retina and the choroid and also perhaps of the inner layers of the sclera along the ora serrata.

In addition, there was in this case a traumatic lesion of the macula such as was described almost simultaneously by Haab, Kuhnt and Ogilvie in 1900. The anatomic substrate of this lesion has been described in the report of the first two of my cases (first section).

It must be noted that in this case the tension remained high notwithstanding the fact that the blood was being resorbed. The tension dropped to a normal level after the removal of the lens. This fact cannot be used to support the theory that the lens caused the high tension, as the beneficial effect of the extraction of the lens might just as well be interpreted in favor of opening the anterior chamber. The rôle of the lens in the causation of traumatic glaucoma will be discussed later.

In the iris loss of structure of the anterior lamella, depigmentation of the posterior lamella and thinness of the whole iris were found. These were also observed on microscopic examination in both my cases reported in the first section. Tears of the iris were also noted in every case. The condition of the iris, therefore, must have been similar in all three cases.

CASE 4.—J. C. S., a manual laborer aged 34, was struck in the right eye on Aug. 21, 1934, by a piece of wire hanging from a passing motor van. Immediately his eyesight was hazy. The next day he had severe pain. His physician found that the anterior chamber contained blood and administered a drop of atropine. Three days later the patient could still count fingers at a distance of about 15 feet (4.6 meters). That night he had severe pain, and in the morning he found that he could not see.

He was examined in the Eye Clinic of the University of Utrecht on August 25. Visual acuity was found to be perception of light only; there was total hyphemia. No perforating wound was seen. The tension was +1/2. The left eye was normal. Visual acuity was 1, with hypermetropia of 0.75 D. The patient was advised to go to the hospital in his home town.

That same day he was admitted to the University Eye Clinic in Amsterdam. It was found that the eyelids and the bulbar conjunctiva were slightly swollen. There was not much ciliary injection, and no wounds were seen. The cornea was smooth (no vesiculation of epithelium was present). The anterior chamber was completely filled with dark blood. The globe was sensitive to touch. The tension was very high, +3. Vision amounted to perception of light only. The left eye was normal. The tension, according to Schiötz, was 75 mm. in the right eye and 20 mm. in the left eye.

Pilocarpine was prescribed.

On the following day the tension was the same. The anterior chamber was then opened inferiorly. A small amount of fluid escaped, but no blood. The pain was not relieved, and the tension remained high. On the same day, therefore, the anterior chamber was opened once more, this time superiorly. The coagulum was washed out with a fine cannula, and a broad iridectomy was performed. Recovery was uneventful.

The patient was dismissed on September 14. The tension was normal. The fundus was seen hazily through remains of hemorrhage into the vitreous. Visual acuity was counting five fingers at 0.5 meter. There was good projection of light. Examination of the visual field showed that the superior quadrants were missing, probably owing to nests of hemorrhage in the vitreous.

On November 14 the patient was seen for the last time. The cornea exhibited a rest of *Durchblutung*. The iris was markedly atrophic; there was a total artificial coloboma superiorly, two anterior synechia inferiorly, a posterior synechia laterally and remains of exudate of the anterior surface of the lens. There were no dislocation and no trembling. The vitreous showed a few coarse and punctate opacities. The optic papilla was normal. The macula exhibited a sharply defined, round, clear area, surrounded by a dull area. Peripherally, pigmentation was seen along the ora serrata, excepting laterally and inferiorly. This pigmentation was fairly coarse and must have been choroidal. Moreover, the pigment was arranged here and there, especially superiorly, along the choroidal vessels, and in one place it was crossed by a retinal vessel. Here and there the pigmentation was relieved by a paler area. Visual acuity was 1/2 after correction with a —1 cylinder. The visual field was normal.

In the sound eye visual acuity was 1.

In this case glaucoma probably commenced on the fourth day. The patient's complaints before that time may be explained by the contusion and by the hyphemia. On the fourth day, however, he had severe pain, and his visual acuity diminished to perception of light, probably owing to a fresh, large hemorrhage. It is difficult to judge the effect of atropine administered on the second day, because in several cases mentioned in the literature this treatment has been carried out without poor results. However, the effect of mydriatics will be discussed later.

Although the tension was high, the corneal surface remained smooth. The peculiar composition of the blood in the anterior chamber is also remarkable. This must be different from the hyphemia that easily escapes with paracentesis after an operation for cataract. This blood cake probably consisted of a mixture of blood and albuminous exudate such as was mentioned by Morax (1921, eighteenth case) or a gelatinous exudate such as was mentioned by other authors, e. g., Schmidt-Rimpler

(1883), Sala (1904, first case) and Cordes and Horner (1933, first and third cases). In my case to be reported next, also, a glutinous fluid was noticed during the operation, while Stoewer (1904) described a blackish-red mass similar to the mass in my case. Wagenmann expressed the opinion that a gelatinous transudate is not uncommon after serious contusion and that it disappears in a short time. In any case it is important to note that no aqueous escaped when the anterior chamber was opened. This is worth remembering.

The iris exhibited an anterior synechia peripherally and marked atrophy of its stroma; both changes were observed also in the cases in which microscopic examination was made. One is also struck by the extensive pigmentation along the ora serrata, probably caused by a choroidal lesion. The lesion of the macula also must have been traumatic.

CASE 5.—C. G., a grocer aged 35, was struck in the right eye by a belt on May 20, 1935. Although his eye was painful, he could see well for two days. During the night he felt a severe pain and sought his physician's advice.

On May 23 there was marked injection of the pericorneal conjunctiva. The cornea was hazy in its middle part; little light was reflected from the retina, and the latter could not be seen. The tension was +1. A bandage was applied.

The tension dropped, but on May 27 the patient had severe pain and vomited. Physostigmine was prescribed, but the tension remained high, i. e., 60 mm. according to the Schiötz scale. Iridectomy was then performed. It was noticed that only a small amount of sticky fluid escaped but that the anterior chamber remained deep. Recovery was uneventful.

On November 26, apart from the total artificial coloboma of the iris, a few opacities were present in the vitreous and a few rests of hemorrhage were observed in its lower part. In the periphery of the fundus, above and laterally and just posterior to the ora serrata the retina was slightly hazy over a small area. This area was surrounded by a small amount of pigment and contained a small horseshoe-shaped tear with a prominent border. No other changes were found, notably, no dislocation or partial dislocation of the lens. The tension was normal. Visual acuity was normal with glasses. The other eye was normal in every respect, being emmetropic and having normal visual acuity.

In this case a fairly young man had an attack of acute glaucoma on the third day after contusion. It was necessary to carry out iridectomy. It is important to note that the iris in this case was normal apart from the artificial coloboma and also that a hemorrhage into the vitreous had occurred.

CASE 6.—A man aged 20 years was struck in the right eye by a cricket-ball on Feb. 1, 1930. He was examined a few hours afterward. The cornea was then clear. There was a slight hyphemia. The pupil was perhaps a little wider than that of the other eye but reacted well to light. Visual acuity was normal in both eyes.

The hyphemia was resorbed in a few days.

On February 6 the patient returned and said that he was seeing colored rings around the lights. The cornea was steamy, the corneal epithelium vesiculated, and the pupil did not react well to light and was abnormally wide. The tension was from +2 to +3. Pilocarpine was prescribed.

The following day the tension was normal and the pupil narrow. The patient was kept under observation for a few days. He then went abroad and was seen no more.

This patient suffered a typical attack of acute glaucoma six days after the accident. It should be noted that the attack occurred after the intra-ocular hemorrhage had been resorbed. This patient responded well to pilocarpine.

CASE 7.—G. B., a rammer aged 54, was struck in the right eye by a large piece of wood on Oct. 25, 1934. His eye was painful immediately; he could see no more and felt sick.

Two hours after the accident he was examined. There were slight abrasions of the eyelids. The conjunctiva was intact. The cornea was steamy, and the

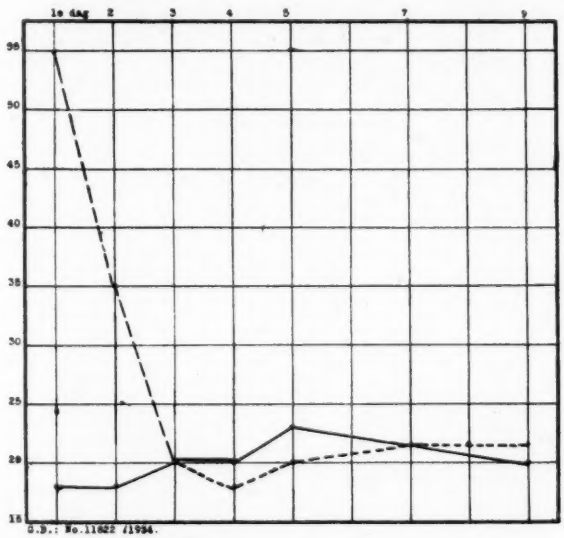

Fig. 23 (case 7).—Diagram showing tension in each eye in the course of traumatic glaucoma of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

pupil fairly wide. No hyphemia and no tears of the iris were present. The fundus was not to be seen. The movements of both eyes were normal. The left eye was normal.

Visual acuity in the right eye was 1/2; there was no correction with glasses.

Visual acuity in the left eye was 1; the eye was emmetropic.

The tension in the right eye, according to the Schiötz scale, was 55; that in the left (normal) eye was 18. Pilocarpine was prescribed. Figure 23 shows the course of the tension of each eye.

The patient was discharged on November 25. The pupil of the right eye was still somewhat wider than that of the other eye and was slightly irregular in shape. There was slight torsion of the fibers of the iris, but the pupillary reaction to light was equal on both sides. Close examination of the fundus revealed no pathologic changes. The tension was normal; visual acuity was normal, and the eye was emmetropic.

In this case glaucoma, associated with a hazy cornea and wide pupil, immediately followed the trauma. This case is remarkable for the absence of any visible traumatic lesions.

The ocular tension was measured with the Schiötz tonometer during nine days. A glance at figure 23 will show that glaucoma was followed by a longer period of hypotony. Also I wish to draw attention to the similarity between this figure and the following figures illustrating the effects of contusion of the eyeball in which only hypertony but no other sign of glaucoma could be found.

CASES OF SIMPLE CONTUSION

It was found (Leplat and Schmidt and de Decker) that in animals contusion is followed by a short period of hypertony (from thirty to forty minutes) and this is followed by a longer period of hypotony (from three to seven days). As it is impossible to take tonometer

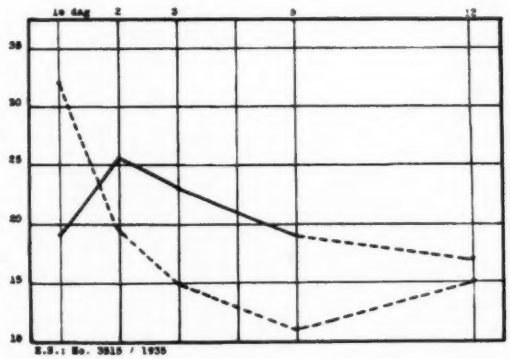

Fig. 24 (case 8).—Diagram showing tension in each eye following simple contusion of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

readings every few minutes on the human subject, one has to be content with daily readings. Even then one is struck by a certain similarity between the curves for animals (compare the original articles, or Schieck and Brückner's "Kurzes Handbuch der Ophthalmologie," volume 4, page 454), the case of traumatic glaucoma just reported and the following cases of simple contusion.

Case 8.—E. S., aged 20, a draughtsman, struck himself in the right eye with the metal end of a tape measure on March 14, 1935. He was examined a few hours later.

The right eye showed superficial erosions of the cornea. The pupil was oval in a superomedial direction and wider than that of the other eye. A fairly large hyphemia was present; the fundus was not to be seen. Visual acuity amounted to perception of movements of the hands.

The left eye was normal, with normal visual acuity.

Refraction showed hypermetropia of 0.5 D. in each eye.

The tension (Schiötz) was 32 mm. in the right eye and 19 mm. in the left (the sound eye).

The following day the cornea was slightly hazy, and its posterior surface was covered with a fine precipitate. The hyphemia had disappeared; the pupil was still oval, and the optic papilla could be fairly well seen. Visual acuity was 1/6. The cornea cleared rapidly, and the reaction of the pupil returned in a few days. On March 18 visual acuity was normal. However, slight paresis of accommodation remained. On March 19 careful examination of the fundus revealed a circumscribed detachment of the retina superiorly and medially, with several small holes.

On April 2 the detachment of the retina had healed spontaneously. On April 9 the patient said that he had no more trouble with reading at a short distance.

CASE 9.—J. A., aged 15, was struck in the right eye with a metal buckle on March 13, 1935. He was examined a few hours later.

The right eye was normal.

Examination of the left eye showed that the eyelids were slightly swollen. The ocular movements were unimpaired. There was a slight pericorneal injection.

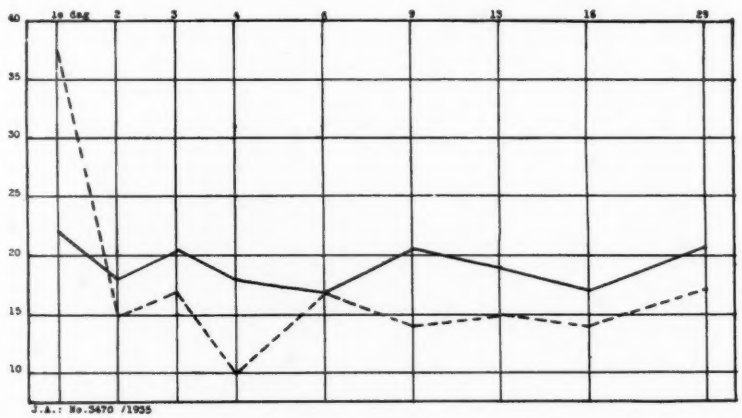

Fig. 25 (case 9).—Diagram showing tension in each eye following simple contusion of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

The cornea was slightly hazy, smooth and not swollen. A large hyphemia was present in about one third of the anterior chamber; the aqueous was slightly turbid. Slight iridodialysis was present medially and superiorly; the pupil of the injured eye was flattened and wider than that of the other eye. There was no reaction of the pupil to light, and no light was reflected from the retina. Visual acuity amounted to perception of movements of the hand.

The tension (Schiötz) was 22 mm. in the right eye (the sound eye) and 37.5 mm. in the left.

Figure 25 shows the course of the tension.

Pilocarpine was prescribed.

On the following day the pupil was narrow; there was a small hyphemia, and the optic papilla was visible. Visual acuity was normal in each eye. Refraction showed hypermetropia of 1 D. in the left eye and of 1.5 D. in the right.

On April 3 the fundus was examined under the influence of a mydriatic. No pathologic changes were noted.

CASE 10.—J. R., a fitter, aged 24, received a blow with a fist on the left eye on April 11, 1935. He was examined a few hours later.

The left eye showed a small tear of the inferior lid and a small subconjunctival hemorrhage. The cornea was clear. The pupil was slightly oval but was equal in size to the other pupil and reacted as well to light. The optic papilla could be well seen and showed no abnormality.

Visual acuity was normal in each eye; refraction showed hypermetropia of 0.75 D. in the right eye and of 0.25 D. in the left. The fundus was normal.

The tension of each eye may be read from figure 26.

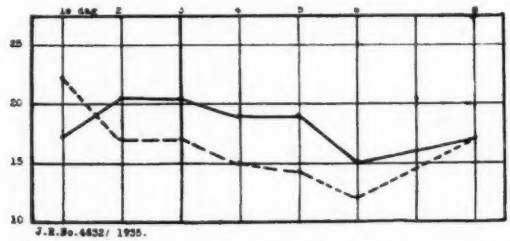

Fig. 26 (case 10).—Diagram showing tension in each eye following simple contusion of the left eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

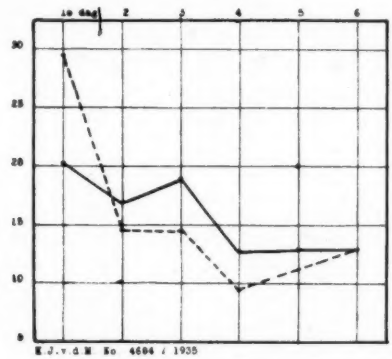

Fig. 27 (case 11).—Diagram showing tension in each eye following simple contusion of the left eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

CASE 11.—K. J. v. d. M., the driver of a car, aged 28, was struck in the left eye by a piece of wood on April 13, 1935. He was examined a few hours later.

The right eye was normal.

The left eye showed a hematoma of the superior lid; the aqueous was turbid, and there was a small blood coagulum in the anterior chamber. The fundus, as far as could be ascertained, was normal.

Two days later visual acuity was normal in each eye. There was a slight degree of hypermetropia (1 D.). The tension of each eye may be read from figure 27.

After six days the pupil was still slightly oval, but it reacted well to light. No pathologic changes could be observed in the fundus.

CASE 12.—M. L., aged 7, was struck on the right eye by a tennis ball on April 26, 1935. He was examined one and a half hours later.

Examination of the right eye showed a slight pericorneal injection. The aqueous was turbid, with a fine keratic precipitate. The pupil, larger than that of the left eye, was slightly oval in a vertical direction and showed a slow reaction to light.

Visual acuity was 1/6 in the right eye and normal in the left; both eyes were emmetropic.

The ocular tension of each eye may be read from figure 28. It must be remembered, however, that the first two readings have only relative value, as the child was frightened and could not be examined well. The figures represent the reading found a few times in succession. Later the patient was calm when he allowed himself to be examined. The course of the ocular tension may be read from figure 28.

On the following day the slight keratic precipitate was still present; the pupil was oval in a horizontal direction, and the pupillary border was slightly damaged on the medial side. The anterior chamber was deeper than that of the sound eye, and the iris trembled, i. e., the lens had been slightly dislocated. The fundus was well seen and showed no pathologic changes.

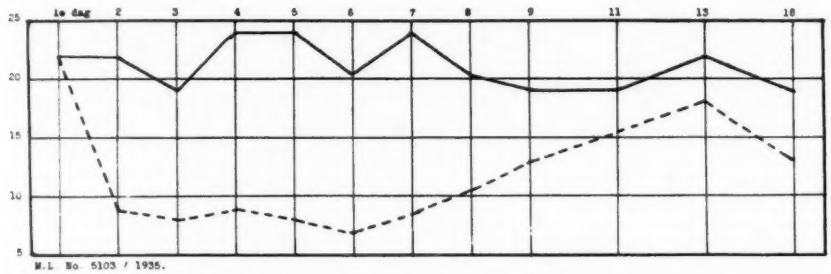

Fig. 28 (case 12).—Diagram showing tension in each eye following simple contusion of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

After eighteen days the pupillary reaction to light returned. Visual acuity was normal.

After this the patient stayed away, so that the fundus was never examined under the influence of a mydriatic. It is possible, therefore, that minor details have been overlooked.

CASE 13.—Nora van V., a girl aged 8, was struck in the left eye by a toy arrow. She was examined within an hour after the accident.

The right eye was normal.

Examination of the left eye showed a small wound of the bulbar conjunctiva medially, with a small subconjunctival hemorrhage. The cornea was a trifle hazy, especially below; its surface, however, was smooth. The iris was greenish. The pupil was round and equal in size to the other pupil but did not react to light.

The child allowed the ocular tension to be measured without any reluctance. The tension was equal on both sides—18 mm. (Schiötz). Figure 29 shows the course of the ocular tension.

Visual acuity was 1/6 in the right eye (the sound eye); with a cylinder of +2.5 D. it amounted to 1/2.

Visual acuity was 1/6 in the left eye, with a cylinder of +3 D. it was 1/2 partly.

This low visual acuity is explained by the fact that this child, although suffering from astigmatism, had never worn spectacles.

After eight days the pupillary reaction to light was normal again.

On May 21 the fundus was examined under the influence of a mydriatic to get a better view of the picture that had been noted before. Medially and inferiorly a sector-shaped pale area was seen, with the broad part of the sector turned to the ora serrata. This area was paler than the surrounding retina and showed scattered fine dots and somewhat coarser pigment dots. The fine dots seemed to be arranged along the choroidal vessels in a few places. The coarser dots, of which only a few were seen, were surrounded by a small yellowish-white ring, and their size corresponded to the size of the choroidal vessels. In one place three of these dots were seen lying in a row in front of a choroidal vessel. One of the choroidal vessels was flanked by yellowish-white stripes.

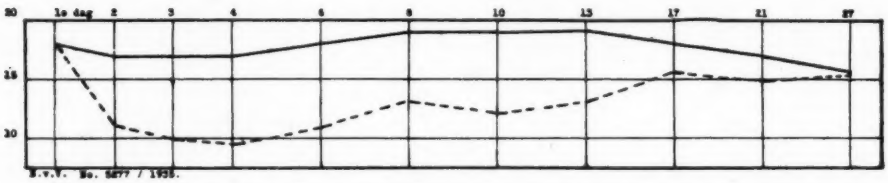

Fig. 29 (case 13).—Diagram showing tension in each eye following simple contusion of the left eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

TABLE 1.—*Tension in Millimeters for Case 14*

Date	Right Eye	Left Eye	Difference
10/24/35	22	17	+5
10/25/35	18	18	0
10/28/35	17	17	0

In the following cases the patients were not examined so often. Each one, however, was seen on the day of the accident. Generally an insignificant contusion of the globe caused the patient to seek advice.

CASE 14.—J. C. B., aged 30, was struck in the right eye by a jet of water. He was examined the next day.

Visual acuity was normal for each eye, with hypermetropia of 1.5 D.

Examination of the right eye showed that the pupil was smaller than that of the sound eye. The reaction to light was good. Otherwise, the eye was normal.

The tension according to Schiötz is shown in table 1.

CASE 15.—J. B., aged 59, was struck in the right eye by a piece of wood. He was examined the same day.

The right eye was emmetropic. The results of tests of visual acuity were not reliable, and there was a large hyphemia.

The left eye had normal visual acuity with a cylinder of +0.5 D.

The tension according to Schiötz is shown in table 2. After that the patient was not seen any more.

CASE 16.—J. W., aged 14, was seen two and one-half hours after contusion of the left eye by a small piece of wood.

The right eye had normal visual acuity, with hypermetropia of 0.5 D.

The left eye had visual acuity of 1/3; vision was normal with a —0.75 sphere. Visual acuity in this eye later returned to normal without glasses.

TABLE 2.—*Tension in Millimeters for Case 15*

Date	Right Eye	Left Eye	Difference
7/31/35	23.0	20.0	+ 3.0
8/ 1/35	19.0	25.5	— 6.5
8/ 2/35	15.0	25.5	—10.5
8/ 5/35	19.0	22.0	— 3.0
8/ 7/35	15.5	19.0	— 3.5

TABLE 3.—*Tension in Millimeters for Case 16*

Date	Right Eye	Left Eye	Difference
7/ 7/35	17.0	16.0	— 1.0
7/ 8/35	19.0	7.5	—11.5
7/10/35	17.0	8.5	— 8.5
7/12/35	15.5	8.5	— 7.0
7/15/35	18.0	8.5	— 9.5
7/17/35	18.0	12.0	— 6.0
7/19/35	20.5	13.0	— 7.5
7/22/35	22.0	14.0	— 8.0
7/26/35	19.0	15.0	— 4.0
7/31/35	19.0	18.0	— 1.0

TABLE 4.—*Tension in Millimeters for Case 17*

Date	Right Eye	Left Eye	Difference
11/16/35	17	25.5	+8.5
11/17/35	17	19.0	+2.0
11/18/35	19	22.0	+3.0

There were slight abrasions of the corneal epithelium. The anterior chamber was deep, and the aqueous was slightly turbid (the next day a small hyphemia was seen). The pupil was slightly contracted and angular and showed no reaction to light. The fundus showed a white spot of Berlin's edema in the upper portion.

The tension according to Schiötz is shown in table 3.

CASE 17.—P. R., aged 38, was seen three or four hours after contusion of the left eye (he struck his head against a bar of iron).

Tests of visual acuity and refraction were not carried out. However, there were no clinical signs of predisposition to glaucoma.

The left eye showed a small tear of the bulbar conjunctiva and erosion of the cornea. Otherwise, there were no changes.

The tension according to Schiötz is shown in table 4.

CASE 18.—H. K. L., aged 26, was seen half an hour after contusion of the left eye by a fragment of brass.

Visual acuity was normal in each eye, and the eyes were emmetropic.

The left eye showed a small wound of the conjunctiva and erosion of the cornea; otherwise, it was normal.

The tension according to Schiötz is shown in table 5.

CASE 19.—J. V., aged 41, was seen one-half hour after contusion of the right eye by a hammer.

Visual acuity of each eye was 1 partly; with a +0.5 sphere it was normal.

The right eye showed a small hemorrhage under the bulbar conjunctiva. The pupil was slightly larger and reacted more slowly to light than that of the left eye. Otherwise, no changes were seen.

The tension according to Schiötz is shown in table 6.

TABLE 5.—*Tension in Millimeters for Case 18*

Date	Right Eye	Left Eye	Difference
12/11/35	17.0	15	—2.0
12/12/35	17.0	12	—5.0
12/13/35	15.5	13	—2.5
12/16/35	13.0	13	Equal

TABLE 6.—*Tension in Millimeters for Case 19*

Date	Right Eye	Left Eye	Difference
12/24/35	15.5	20.5	—5
12/27/35	15.5	20.5	—5

TABLE 7.—*Tension in Millimeters for Case 20*

Date	Right Eye	Left Eye	Difference
1/ 9/36	27.5	27.5	Equal
1/10/36	27.5	19.0	—8.5
1/11/36	27.5	24.0	—3.5
1/13/36	27.5	20.5	—7.0

CASE 20.—W. G. O., aged 7, was seen one hour after contusion of the left eye by a stick.

On the second day after the accident visual acuity was normal in each eye, with hypermetropia of 1 D.

The left eye showed (on the day of the accident) erosion of the cornea; otherwise, there were no changes.

The tension according to Schiötz is shown in table 7.

CASE 21.—P. V., aged 16, was seen one-half hour after contusion of the left eye by a fragment of iron.

Visual acuity was normal; there was hypermetropia of 0.5 D.

Examination of the left eye revealed erosion of the cornea. The pupil was slightly oval and was slightly more contracted than the pupil of the other eye. Otherwise, no changes were seen.

The tension according to Schiötz is shown in table 8.

CASE 22.—H., aged 37, was seen one-half hour after contusion of the left eye by an accidental kick.

The left eye showed a small wound of the eyelids and erosion of the cornea. The pupil was obliquely oval and showed only a slight reaction to light. Temporally and above, Berlin's edema of the retina was seen. There were no tears and no hemorrhage.

On the following day visual acuity was normal in the right eye (the eye was emmetropic); visual acuity in the left eye was 1/2, and with a +0.5 D. cylinder it was normal.

The tension according to Schiötz is shown in table 9.

TABLE 8.—*Tension in Millimeters for Case 21*

Date	Right Eye	Left Eye	Difference
1/10/36	20.5	15.5	−5
1/11/36	20.5	15.5	−5
1/17/36	20.5	20.5	Equal

TABLE 9.—*Tension in Millimeters for Case 22*

Date	Right Eye	Left Eye	Difference
1/22/36	15.5	18.0	+2.5
1/23/36	20.5	15.5	−5.0
1/24/36	20.5	17.0	−3.5

COMMENT

Summarizing the clinical cases of traumatic glaucoma, one finds the following:

In the third case there were subluxation of the lens, a torn and atrophied iris, a hemorrhage into the vitreous, a hole at the macula and a rupture of the choroid and the retina close to the ora serrata. Visual acuity was movements of the hands only.

In the fourth case there were hemorrhage into the anterior chamber and the vitreous, atrophy of the iris with peripheral anterior synechiae, rupture of the choroid and retina near the ora serrata and a slight injury of the macula lutea. Visual acuity was 1/3.

In the fifth case the aqueous was sticky, and there were a hemorrhage into the vitreous and peripheral rupture of the retina and perhaps also of the choroid. Visual acuity was normal.

In these cases it was necessary to operate. The result was satisfactory, as the impaired visual acuity in the third and fourth cases is explained by the injury to the macula lutea. It will be noted that in all these cases a hemorrhage into the vitreous had occurred, in one case combined with dislocation of the lens. In the following cases these serious injuries were absent, and conservative treatment gave satisfactory results.

In the sixth case there was an attack of acute glaucoma.

In the seventh case there was an attack of acute glaucoma without visible injuries to the inner part of the eye. The curve for tension of this eye shows great similarity to the curves in the cases of simple contusion, in most of which the eye showed slight injuries.

Summarizing the cases of simple contusion, one finds that in fifteen cases slight intra-ocular lesions and a varying degree of instability of tension were present. In case 12 hypotony was combined with subluxation of the lens. In the literature dislocation of the lens is regarded as one of the most important causes of glaucoma associated with dislocation of the lens. This theory is erroneous, as will be discussed later (see "Conclusions"). Instability of tension without any accompanying visible intra-ocular injury (in a few instances in this series the pupil

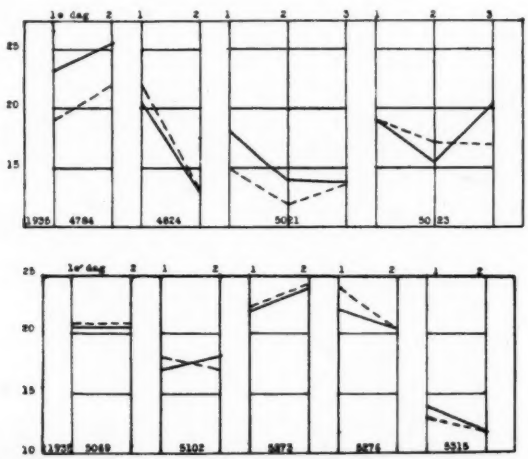

Fig. 30.—Diagrams showing tension in each eye in nine control cases. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

was slightly larger than that in the sound eye) was observed in seven cases (10, 14, 17, 18, 19, 20 and 21). In case 14 (the patient was examined on the second day) and in case 17 (the error of refraction was unknown) a few details are missing, but in the remaining five cases the clinical examination was complete.

On comparing all the cases of traumatic glaucoma (anatomic and clinical) one finds that in two cases (1 and 2) severe intra-ocular lesions were present, and the eye had to be removed (see "Anatomic Part"); that in two other cases (3 and 4) severe lesions were present but the eye was saved, although visual acuity was reduced; that in yet two other cases (5 and 6) the lesions were much less severe, and vision returned to normal. In the last case (7) there was no intra-ocular lesion. It is remarkable that the curve for tension in this case resembles the curves found in cases of simple contusion.

In the cases of simple contusion slight injuries were found in one group and no injuries in another. Yet in all the cases there was instability of tension.

From this comparison one may conclude that from the most severe cases of traumatic glaucoma to the simplest cases of contusion every intermediate stage is found in two respects: (*a*) as regards the severity of the intra-ocular injury and (*b*) as regards the severity of the instability of tension.

One may also conclude that instability of tension may occur in young patients with normal eyes after contusion causing no visible injury to the inner part of the eye.

As it seemed possible that the dressing applied to the eye might influence the intra-ocular tension, nine control cases were observed. No marked influence could be ascertained.

III. ANALYSIS OF CASES REPORTED IN THE LITERATURE

In this chapter all the cases of traumatic glaucoma reported in the available literature are considered, together with the cases described in the two previous sections. Other cases have been added which were not described as instances of traumatic glaucoma but which may be regarded as such.

These cases were grouped according to the accuracy of the description as follows:

I. Reliable cases (refraction between hypermetropia of 3.5 D. and myopia of 10 D.).

II. Less reliable cases (refraction not known).

The reliable cases and the unreliable cases were grouped according to the result of treatment as follows:

(*a*) Cases in which the result of treatment was favorable: tension normal; visual acuity 1/2 or more.

(*b*) Cases in which the result of treatment was less favorable: tension normal; visual acuity from 1/60 to 1/3.

(*c*) Cases in which the result of treatment was unfavorable: tension raised or visual acuity less than 1/60 or eye removed.

The old figures indicating errors of refraction were recalculated in diopters, and the figures indicating visual acuity were reduced to simple fractions.

The third and fourth groups are unsuitable for study and may be dismissed without discussion.

III. Cases without (sufficient) obtainable data. These were reported by the following authors: Aubineau (1910), Chalupecky (1905),

TABLE 10.—*Data for Reliable Cases in Which the Result of Treatment Was Favorable (Group I a)*

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes					Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result*	Comment
				Hemorrhage	Iris	Lens	Retina	Choroid	Glaucoma	First Treatment				
Bralley, 1883	14	Cork	Hypermetropia −2	.....	.....	Cataract	.....	.....	2 days	8 days	More than 20 days	Physostigmine	T.N.; V. = 0.5	
Ferber, 1887	?	Piece of iron	Hypermetropic astigmatism −2	.....	.....	.....	Hemorrhage and exudation	.....	.....	3 wks.?	9 wks.	Physostigmine; iridectomy	T.N.; V. = 0.5 (partly)	
Knapp, 1883	29	Stick	Emmetropia?	.....	.....	Anterior dislocation	.....	.....	12 days	A few days	1 day	Physostigmine	T.N.; V. = 1 with glasses	At first hypotony; later, hypertony, with normal anterior chamber and turbid vitreous
Lavat, 1924	41	Fist	Hypermetropic astigmatism −1.5	Corpus vitreum	.....	.....	.....	.....	?	18 days	5 mos.	Pilocarpine; iridectomy; trepanation; paracentesis	T.N.; V. = 1 with glasses	Optic nerve cupped
Magitot, 1917, first case	34	Explosion	Emmetropia?	.....	.....	.....	.....	.....	?; in both eyes	1 day	3 days	.....	T.N.; V.: Right eye, ⅔; left eye,1	
Magitot, 1917, second case	?	Fall	Myopic astigmatism +0.5	.....	.....	.....	Slight venous dilatation	.....	?	? hrs.	5 days	.....	T.N.; V. = ⅔ with glasses	
Magitot, 1917, third case	32	Indirect	Emmetropia?	.....	.....	.....	.....	.....	?	4 days	8 days	.....	T.N.; V. = 1	
Magitot, 1917, fourth case	?	Lump of clay	Myopia +1	Anterior chamber	.....	.....	.....	.....	?	5 days	6 days	.....	T.N.; V. = 1 with glasses	
Magitot, 1918, first case	24	Fall	Emmetropia?	.....	.....	.....	.....	.....	?; in both eyes	2 days	8 days	.....	T.N.; V. = 1	Fracture of orbit
Marx and Quartero, 1930	13	Piece of coal	Myopic astigmatism +0.75	Anterior chamber	.....	Trembling	.....	Tears; ablatio	21 days	.....	About 4 wks.	Atropine; pilocarpine	T.N.; V. = 1 with glasses	At first, hypotony; atropine discontinued on twenty-first day
Motolese, 1912	?	?	Hypermetropia +2.5	.....	.....	Anterior chamber shallow	.....	.....	?	?	?	Physostigmine	T.N.; V. = 0.6	

Myers, 1901.....	?	Stone	Astigmatism	Anterior chamber; corpus vitreum	.....	Subluxation	Commotio	Subretinal hemorrhage	5 days	.....	7½ wks.	Atropine; physostigmine; pilocarpine	T.N.; V. = 1 with glasses	Atropine replaced by miotics on fifth day because of slightly raised tension; in third week attack of acute glaucoma with deep anterior chamber
Peters, 1904, first case	29	Piece of wood	Hypermetropia —1	.....	.....	Cataract	.....	.....	?	18 days	4 wks.	Physostigmine	T.N.; V. = 5/6 with glasses	
Peters, 1904, second case	57	Whip	Emmetropia?	Anterior chamber	.....	.....	.....	.....	.....	.....	7 days	Atropine; physostigmine; paracentesis	T.N.; V. = 5/7	Atropine administered during 8 days
Pye-Smith, 1882.....	70	Fall	Hypermetropia —1.5	?	?	?	?	?	3 days?	5 days	21 mos.	Physostigmine	T.N.; V. = 1 (partly)	
Sala, 1904, first case....	53	Cork	Hypermetropia —1.25	Anterior chamber; corpus vitreum	.....	.....	?	?	.....	.....	3 mos.	Paracentesis	T.N.; V. = 5/7	
Sala, 1904, second case	38	Stone?	Hypermetropia —3.5	Anterior chamber	.....	.....	.....	.....	5 days	.....	6 wks.	Scopolamine; physostigmine; paracentesis	T.N.; V. = 1	Because of hypotony scopolamine given until fifth (?) day
Scheffels, 1891.....	35	Stick	Myopia 1⅔	Anterior chamber; corpus vitreum	Dialysis	Cataract	Detachment	.....	.....	2 days	12 wks.	Atropine; physostigmine	T. + ?; V. = ⅔	After 12 years tension normal; total cataract; visual acuity 1/60
Tillema, fifth case.....	35	Belt	Hypermetropic astigmatism —0.5	Corpus vitreum	.....	.....	Small tear	.....	3 days	3 days	7 days	Physostigmine; iridectomy	T.N.; V. = 1 with glasses	Anterior chamber remains deep after having been opened
Tillema, seventh case...	54	Piece of wood	Emmetropia	.....	.....	.....	.....	.....	.....	Hours	2 days	Pilocarpine	T.N.; V. = 1	
Villard, 1905, first case	66	Piece of wood	Hypermetropia —1	.....	.....	.....	.....	.....	.....	12 days	4 days	Physostigmine	T.N.; V. in right eye and left eye, 1/3	
Villard, 1906, third case	63	Wooden stopper	Myopia +5	Anterior chamber	Tear	Trembling	.....	.....	.....	4 hrs.	4 days	Physostigmine; epinephrine; pilocarpine	T.N.; V. = 0.3 with glasses	Other eye: visual acuity with glasses, 0.4

* T. indicates tension; T.N., normal tension, and V. vision.

TABLE 11.—*Data for Reliable Cases in Which the Result of Treatment Was Less Favorable (Group I b)*

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes					Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result*	Comment
				Hemorrhage	Iris	Lens	Retina	Choroid	Glaucoma	First Treatment				
Agnello, 1931, second case	75	Branch of tree	Emmetropia	?	?	?	?	?	3 days	3 mos.	?	Iridectomy	T.N.	
Bourgeois, 1904, first case	32	Cork	Emmetropia	Anterior chamber	.....	Anterior subluxation	.....	.....	1 day	1 day	3 mos.	Pilocarpine	T.N.; V. = 0.25 with glasses	Traumatic myopia of 3.5 D.
Brand, 1905.....	30	Fragment of steel	Hypermetropia —2.5	.....	.....	.....	.....	.....	5 days	.....	2 mos.	Atropine; scopolamine; physostigmine; iridectomy	T.N.; V. = 1/3	Phystostigmine administered when optic nerve was already cupped
Darier, 1899.....	46	Piece of wood	Emmetropia?	?	?	Anterior chamber very shallow	?	?	4 wks.	A few days?	?	Physostigmine; iridectomy	T.N.; V. = 1/3 with glasses	Traumatic myopia of 3.5 D.; other eye emmetropic, with visual acuity of 1
Fromaget, 1913, second case	25	Piece of wood	Hypermetropic astigmatism —1.5	Anterior chamber	.....	Trembling	.....	.....	.....	2 hrs.	3 wks.	Pilocarpine	T.N.; V. = 1/3	
Herrmann, 1906.....	47	Iron nut	Hypermetropia —2.25	Corpus vitreum?	.....	Cataract; anterior chamber shallow	?	?	.....	4 days	4 days	Physostigmine; iridectomy	T.N.; V. = 4/20	
Pick, 1904.....	40	Jet of water	Emmetropia	Corpus vitreum	.....	Cataract	.....	Equatorial choroiditis	Weeks or months	1 yr.	2 wks.	Pilocarpine; iridectomy	T.N.; V. = 1/5	Shallow, gray excavation of optic nerve
Stoewer, 1904.....	23	Stick	Emmetropia?	Anterior chamber; corpus vitreum	.....	Cataract	.....	Small scar	4 days	2 days	7 mos.	Procaine borate; pilocarpine; physostigmine; sclerotomy twice; iridectomy	T.N.; V. = 4/20	Optic nerve slightly atrophic
Stoewer, 1907.....	65	Explosion	Hypermetropia —3.5	?	?	?	?	?	?	?	?	Iridectomy	T.N.; V. = 4/25	Central old macula corneae

* T. indicates tension; T.N., normal tension, and V. vision.

TABLE 12.—*Reliable Cases in Which the Result of Treatment Was Unfavorable (Group 1 c)*

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes					Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result*	Comment
				Hemorrhage	Iris	Lens	Retina	Choroid	Glaucoma	First Treatment				
Tillema, fourth case...	34	Brass wire	Hypermetropia —0.5	Anterior chamber; corpus vitreum	Atrophy	.....	Macular lesion	Peripheral tear	4 days	1 day	1 day	Atropine; pilocarpine; paracentesis; iridectomy	T.N.; V. = ⅓ with glasses	
Agnello, 1931, first case	54	Fist	Hypermetropic astigmatism	?	?	?	?	?	10 days	8 days	?	Miotics; iridectomy	T.+; enucleation elsewhere	
Magitot, 1917, fifth case	41	Branch of tree	Emmetropia	Anterior chamber	Tear	.....	.....	.....	1 day?	2 days?	6 wks.	Pilocarpine; physostigmine; paracentesis	T.N.; V. = perception of light	Fracture of orbit
Priestley Smith, 1881...	57	Cork	Hypermetropia —2.5	?	Detachment	Subluxation?	?	?	.....	9 wks.	3 days	Physostigmine	T.+2; V. = 0; enucleation	
Priestley Smith, 1883...	59	Fist	Hypermetropia —1.5	Corpus vitreum	.....	Temporal subluxation	Not visible	Not visible	3 days	6 days	14 wks.	Physostigmine; iridectomy	Enucleation	Immediately after iridectomy, enucleation because of prolapse of vitreous
Tillema, first case.....	26	Fall	Emmetropia	Anterior chamber; corpus vitreum	Tear	Cataract	Tear	Tear	25 days	Hours	3 wks.	Pilocarpine	T.+; V. = perception of light; enucleation	
Tillema, second case...	30	Piece of wood	Emmetropia	Corpus vitreum	Tear	Capsule torn; luxation	Tear	Tear	7 days?	Hours	7 wks.	Pilocarpine	T.+; V. = perception of light; enucleation	Patient refused regular treatment
Tillema, third case.....	41	Piece of brass	Hypermetropia —0.5	Anterior chamber; corpus vitreum	Tear	Subluxation	Tear; hole at macula	Tear	6 days	Hours	18 days	Pilocarpine; extraction of lens	T.N.; V. = 2/200	

* T. indicates tension; T.N., normal tension, and V. vision.

TABLE 13.—*Data for Less Reliable Cases in Which the Result of Treatment Was Favorable (Group II a)*

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes					Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result*	Comment
				Hemorrhage	Iris	Lens	Retina	Choroid	Glaucoma	First Treatment				
Armaignac, 1907.....	?	Piece of wood	?	?	?	?	?	?	?	?	?	Operation	Complete cure	
Augstein, 1904.....	?	?	?	?	?	?	?	?	?	?	?	Physostigmine	Favorable	
Beauvois, 1908.....	?	?	?	?	?	?	?	?	?	?	?	Iridectomy	T.N.	
Boucheron, 1889.....	Young	Snowball	?	Corpus vitreum	.....	.....	.....	.....	?	8 days	3 days	Paracentesis six times	T.N.; V. = same in both eyes	
Cantonnet, 1912.....	49	Piece of concrete	?	.....	.....	.....	.....	.....	?	1 day	5 days	Cocaine; pilocarpine; atropine physostigmine	T.N.; V. = 1	Atropine with care for tension of —2
Cohn, 1904.....	?	?	?	?	?	?	?	?	?	?	?	Iridectomy	Favorable	
Cramer, 1931, second case	42	Piece of iron	?	Anterior chamber	?	?	?	?	2 days	?	?	Paracentesis	T.N.	
Fromaget and Fromaget, 1913, first case	35	Iron chain	?	Anterior chamber; corpus vitreum	.....	.....	.....	.....	14 days?	.....	?	Pilocarpine; physostigmine iridectomy	T.N.	
Fromaget, 1924.....	?	?	?	?	?	?	?	?	?	?	?	Miotics; epinephrine and procaine hydrochloride retrobulbarly	Favorable	
Guibert, 1922, first case	64	Cork	?	Anterior chamber	?	?	?	?	?	?	14 days	Pilocarpine; sodium iodide subconjunctivally	T.N.; V. = ⅔	Optic nerve paler than that in other eye
Guibert, 1922, second case	58	?	?	?	?	?	?	?	?	18 days	14 days	Pilocarpine; sodium iodide subconjunctivally	T.N.; V. = 1	
Guibert, 1922, third case	59	?	?	?	?	?	?	?	?	?	2 days	Pilocarpine; sodium iodide subconjunctivally; iridectomy	Favorable	
Jacobson, 1884, first case	67	Wooden stopper	?	Corpus vitreum?	.....	Anterior chamber shallow; cataract	?	?	?	8 days	2 days	Atropine; paracentesis	T.N.; V. = ⅔	Paracentesis = unfinished iridectomy

Loschetschnikov, 1907	?	?	?	?	?	?	?	?	?	?	?	Physostigmine	Favorable	
Loschetschnikov, 1907	?	?	?	?	?	?	?	?	?	?	?	Physostigmine; benzylmorphine hydrochloride	Favorable	
Magitot, 1918, second case	25	Fall	?	.....	.....	.....	.....	.....	13 days	18 days	More than 2 months	Pilocarpine; epinephrine; cocaine	T.N.; V. = 1 (partly)	Fracture of orbit
Mendel, 1909	32	Stick	?	?	?	Anterior subluxation	?	?	4 days	3 days	15 days	Pilocarpine; extraction of lens	T.N.; V. = 0.5 with glasses	Large partial excavation of optic papilla
Morax, 1921, fifteenth case	16	Stone	.....	.....	Dialysis	.....	.....	.....	2 days	?	2 mos.	Pilocarpine	T.N.; V. = 0.8	
Rust, 1903.....	31	Elbow	?	?	?	?	?	?	?	A few days	A few days	Physostigmine; iridectomy	T.N.; V. = 1	
Šafář, 1930.....	?	?	?	?	Tear	Subluxation	?	?	?	?	?	Extraction of lens	T.N.; favorable	
Priestly Smith, 1881..	60	?	?	?	?	?	?	?	?	5 days	3 days	Physostigmine	T.N.; V. = 1 (with reading of Jaeger's test type)	
Stölting, 1913, first case	Young	Snowball	?	.....	.....	.....	.....	.....	?	Hours	1 day	Hot compresses	T.N.; V. = 1	
Stölting, 1913, second case	Young	Horse's bit	?	Anterior chamber	.....	.....	.....	.....	.....	1 hour	1 day?	Physostigmine	T.—?; V. = same in both eyes; intact	
Stölting, 1913, third case	Old	Spectacles	Hypermetropia?	?	?	?	?	?	?	?	A few days	?	T.N.	Tension normal up to patient's death
Terson, 1907, first case	Young	?	?	?	?	?	?	?	?	?	?	Miotics	Favorable	
Terson, 1907, second case	Young	?	?	?	?	?	?	?	?	?	?	Miotics	Favorable	
Terson, 1907, third case	Young	?	?	?	?	?	?	?	?	?	?	Sclerotomy; iridectomy twice	T.N.	
Tillema, sixth case	20	Cricket-ball	?	Anterior chamber	.....	.....	.....	.....	5 days	2 hrs.	1 day	Pilocarpine	T.N.; V. = 1	
Villard, 1906, first case	63	Piece of wood	?	.....	.....	.....	.....	.....	.....	7 days	7 days?	Physostigmine; pilocarpine; epinephrine	T.N.	Patient not seen after seventh day; after 9 months letter: "Visual acuity very satisfactory"
Villard, 1906, second case	39	Piece of wood	?	.....	.....	Subluxation?	.....	.....	?	14 days	5 days	Physostigmine; pilocarpine	T.N.; V. = 0.8	

* T. indicates tension; T.N., normal tension, and V. vision.

TABLE 14.—*Data for Less Reliable Cases in Which the Result of Treatment Was Less Favorable (Group II b)*

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes: Hemorrhage	Iris	Lens	Retina	Choroid	Period Between Trauma and: Glaucoma	First Treatment	With Treatment Equilibrium Returned After	Treatment	Result*	Comment
Bourgeois, 1904, second case	69	Fall	?	.....	.....	Anterior dislocation	.....	.....	?	5 wks.	4 wks.	Pilocarpine; iridectomy	T.N.; V. = 1/6 with glasses	Traumatic myopia of 3 D
Cordes and Horner, 1932, first case	18	Tomato	?	Anterior chamber; corpus vitreum	.....	.....	.....	.....	5 days	?	?	Ethylmorphine hydrochloride; atropine; physostigmine; paracentesis	T.N.; V. = 0.4 with glasses	Patient discharged on fourth day: hemorrhage into vitreous and glaucoma on fifth day after playing football
Hirschberg, 1883, first case	15	Bits of wood and iron	?	Corpus vitreum	Tear	Subluxation	Macular lesion	Tear	7 days	3 wks.	6 wks.	Atropine; physostigmine; iridectomy	T.N.; V. = 15/200	Atropine not administered by Hirschberg
Jacobson, 1884, second case	64	Piece of wood	?	.....	.....	Luxation	Halo glaucomatosus	.....	4 days	4 wks.	?	Paracentesis	T.N.; V. = 0.1	Paracentesis-unfinished iridectomy
Mellinger, 1899	26	Iron chain	?	?	?	?	?	?	Several days	?	?	Iridectomy	T.N.; V. = reduced	
Müller, 1895	44	Branch of tree	?	Corpus vitreum	.....	Luxation in corpus vitreum	?	?	?	14 days	7 days	Physostigmine; paracentesis	T. N.?; V. = 2/60 with glasses	Paracentesis-unfinished extraction of lens
Padovani, 1929	54	Stone chip	?	?	?	?	?	?	30 days	?	?	Pilocarpine	T. lower (normal?)	
Schindhelm, 1917 first case	41	Cow's horn	?	Anterior chamber	Tear	Cataract	?	?	-?	3 days?	16 days?	Physostigmine	T.N.; V. = 5/12?	
Siegfried, 1896	18	Iron nut	?	Anterior chamber; corpus vitreum	.....	.....	Hemorrhage	.....	.....	2 hrs.	?	?	T.N.?; V. = ability to read Jaeger's test type no. 15	Gelatinous exudate in anterior chamber
Villard, 1905, third case	6	Toy pistol	?	Anterior chamber	-?	-?	Detachment	-?	4 days	7 days	4 days	Physostigmine; pilocarpine; ethylmorphine hydrochloride	T.N.; V. = 0.1	Detachment of retina cured; atrophy of optic nerve
Spencer Watson, 1880....	65	Stone	?	Anterior chamber	.....	Trembling	?	?	?	10 days	12 days	Physostigmine; sclerotomy	T.N.; V. = ability to read Jaeger's test type no. 20	

* T. indicates tension; T.N., normal tension, and V. vision.

Fischer (1908), Genet (1922 and 1925), Grand-Clément (1911), Hansell (1902), Helberg (1906), Klauber (1922), Krienes (1895), de Schweinitz (1909) and Wagenmann (1915).

IV. Cases in which predisposition to glaucoma seemed likely (because of hypermetropia); cases in which there was myopia of more than 10 diopters or other abnormalities. These were reported by the following authors: Agnello (1931), Agnew and Webster (1886), Bourgeois (1904), Burchhardt (1896), Deutschmann (1916), Glauning (1902), von Hippel (1900), Landesberg (1869), Mazza (1907), Oliver (1896), Reid (1929), Salzer (1915), Scheer (1903), Schindhelm (1917), Schmidt-Rimpler (1883), Sédan (1925) and Thilliez (1906).

COMMENT ON CLINICAL DATA

The total number of one hundred and six cases, to which may be added a certain number without sufficient data, is small for the number of years over which they have been collected. One must not be too dogmatic, therefore, in drawing conclusions. One would be inclined, for instance, to think that in the majority of cases the course is favorable. This would be an erroneous view, as few cases in which there was dislocation of the lens have been included in the tables, while experience has shown that in such cases the course is usually unfavorable. On the other hand, the tables probably also give a wrong impression of the cases in which the course was favorable because many cases in which the intra-ocular tension was only slightly raised must have escaped notice. My cases of "simple contusion" have convinced me that the intra-ocular tension must be checked tonometrically to find a difference in tension between the two eyes.

Traumatic glaucoma apparently occurs at every age. The extremes are 6 years (Villard, 1905, third case) and 75 years (Agnello, 1931, second case). This is easily understood, as beyond these age limits one is not so much exposed to trauma.

The objects that cause glaucoma vary greatly in size and in weight. A common feature, however, is the fact that they strike the eye with great velocity (e. g., the fist, a cricket-ball).

In seventy-four cases the time of onset of the attack was mentioned. In thirty-two of these the attack came on immediately after contusion; in twenty-one other cases, after from three to five days, and in six cases, a little later, but within the first week. In fifty-nine cases, therefore, glaucoma occurred in the first week. In only six cases did it occur in the second week. In two cases it occurred in the third week, and in the remaining cases still later, up to seven weeks. One may say, therefore, that in the majority of cases glaucoma occurs in the first week after contusion.

TABLE 15.—*Data for Less Reliable Cases in Which the Result of Treatment Was Unfavorable (Group II c)*

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes: Hemorrhage	Iris	Lens	Retina	Choroid	Period Between Trauma and: Glaucoma	First Treatment	With Treatment Equilibrium Returned After	Treatment	Result*	Comment
Augstein, 1904	?	?	?	?	?	?	?	?	?	?	?	Iridectomy	Poor	
Buchanan, 1907	59	Broomstick	?	?	?	Dislocation	?	?	?	?	6 mos.	.....	Enucleation	Partial rupture of sclera
Cabannes, 1908, first case	38	Book	?	.....	.....	Subluxation	?	?	3 days	?	Months	Sclerotomy twice; pilocarpine; iridectomy impossible	T.+2; V. = ?	
Cabannes, 1908, second case	60	Cork	?	Anterior chamber	Tear	.....	?	?	?	?	?	Iridectomy	T.N.; V. = almost blind	Six months later, cataracta traumatica
Cordes and Horner, 1932, third case	35	Whip	?	Anterior chamber	.....	?	?	?	4 days	?	More than 13 days	Atropine; paracentesis twice	V. = 0	
Cramer, 1931, third case	16	?	?	Anterior chamber; corpus vitreum?	?	?	?	?	6 days	?	?	Paracentesis	T.N.; V. = ?	Two months later, detachment of retina
Cramer, 1931, fourth case	25	Iron chain	?	?	?	?	?	?	5 days	?	6 mos.	Paracentesis	T. = 30; V. = 0	Patient did not follow instructions
Cramer, 1931, fifth case	61	Piece of wood	?	?	?	?	?	?	-?	?	Months	Iridectomy; miotics; trepanation; paracentesis	T.N.; V. = 0	
Cramer, 1931, sixth case	59	Piece of wood	?	?	?	Subluxation	?	?	?	?	?	1% physostigmine in live oil; iridectomy	T. = 50; V. = 1/3	
Erdmann, 1900.....	66	Piece of wood	?	?	?	Anterior dislocation	?	?	-?	4 wks.	3 days	.....	V. = perception of light; enucleation	
Fromaget, 1907	35	Iron chain	?	?	?	?	?	?	14 days	Hours	More than 5 wks.	Atropine; pilocarpine; physostigmine; iridectomy	T.+; V. = 1/10?	Atropine discontinued on sixth day; rupture of sclera

Garnier, 1891	14	Stick	?	Corpus vitreum	.....	Trembling	?	?	3 days	.....	12 days	Atropine; physostigmine	Enucleation	Atropine discontinued on third day; partial rupture of sclera?
Goldzieher, 1916	?	Fall?	?	.....	?	Cataract	Fine peripheral pigmentation	.....	2 mos.?	?	?	?	T. = from 40 to 50	Optic nerve deeply cupped
Hirschberg, 1883, second case	38	Piece of iron	Myopia?	.....	.....	?	Hemorrhagic infiltration	.....	3 mos.	A few days	?	Atropine; physostigmine; iridectomy refused	T.+1	Immediately after contusion T.—1; atropine administered; patient dismissed on second day
Hoor, 1888	?	Piece of iron	?	Corpus vitreum?	?	?	Detachment	?	?	?	More than 3 mos.	Pilocarpine; physostigmine; iridectomy	T.+	
Lamb, 1927	9	Rope	?	Anterior chamber	Tear	Cataract	?	?	?	?	8 wks.	?	Enucleation	
Martin, 1907, 3 cases....	?	Cow's horn	?	?	?	?	?	?	?	Av. already = 0	.....	.....	Glaucoma absolutum	
Mezzatesta, 1929.....	76	Fist	?	?	?	?	?	?	3 days	?	?	.....	T.+; V. = 0	
Morax, 1921, seventeenth case	27	Fist	?	.....	.....	Cataract	.....	.....	A few days?	2 yrs.	20 mos.	Extraction of lens; trepanation; sclerectoiridectomy	T. = 10; V. = 0	
Morax, 1921, eighteenth case	65	Jet of water	?	Anterior chamber	?	Anterior dislocation	Hemorrhage	Hemorrhage	1 day	1 day	4 wks.	Pilocarpine; physostigmine; anterior sclerotomy	V. = 0; enucleation	
Scalzitti, 1927	19	Tennis ball	?	?	?	?	Detachment	Pigmentation	30 days	?	?	Miotics; repeated paracentesis	T.N.; A. = 0	
de Schweinitz, 1898.....	46	?	?	?	?	?	Papillitis	?	From 7 to 22 days	7 days?	More than 3 mos.	Physostigmine	T. = + 1; V. = 0	Patient did not follow instructions
Thibert, 1920	33	Fragment of wood	?	?	?	?	?	?	12 or 13 days	13 days?	More than 2 mos.	Pilocarpine	T. = 65	
Villard, 1905, second case	52	Piece of concrete	?	-?	-?	-?	-?	-?	-?	10 days	?	Physostigmine	T.+; V. = 0	Patient refused operation

* T. indicates tension; T.N., normal tension, and V. vision.

In checking the percentage of intra-ocular lesions, tears of the iris and intra-ocular hemorrhage have been grouped together, as a tear of the iris is always accompanied by hemorrhage. Pathologic changes of the choroid and the retina have also been grouped together, as they usually accompany each other. Many cases were found to lack so many details that it seemed inadvisable to use them. In table 16 only the data for the reliable cases have been used, and among these only those cases have been considered in which the required item was reported. Thus this table gives an idea of the relation between the course of the disease and the intra-ocular lesions.

It is found, therefore, that the number of intra-ocular hemorrhages, dislocations of the lens and pathologic changes in choroid and retina run parallel with the course and severity of the disease. Unfortunately,

TABLE 16.—*Relation Between Course and Intra-Ocular Lesions*

	Incidence	Percentage
Hemorrhage		
23 cases with favorable course	12	50
8 cases with less favorable course	6	75
6 cases with unfavorable course	6	100
Dislocation of the lens		
23 cases with favorable course	5	22
8 cases with less favorable course	3	37
6 cases with unfavorable course	4	66
Changes in the choroid and retina		
22 cases with favorable course	7	33
6 cases with less favorable course	3	50
4 cases with unfavorable course	3	75

the number of cases is too small to give these figures absolute value, but it supports my opinion, which will be discussed later, that dislocation of the lens is accompanied by other severe intra-ocular damage and constitutes a symptom of the disease but not its cause. If the course is unfavorable in cases in which there is dislocation of the lens, one may conclude from the dislocation of the lens that other serious lesions are present. The dislocation of the lens possesses, therefore, only prognostic significance.

It was found impossible to draw conclusions from the length of the period that elapsed between the onset of the disease and the first treatment, as the figures are too much influenced by the data for one or two particular cases.

IV. CONCLUSIONS

ORIGIN—PROGNOSIS—TREATMENT

ORIGIN

Although reports of reliable cases of traumatic glaucoma were published as early as 1881 (Priestley Smith), few more than a hundred instances can be collected from the literature. In many other cases

the condition has been labeled "glaucoma through dislocation of the lens," a term which is attractive but misleading, as will be discussed later. Anatomically, little was known about traumatic glaucoma as the eye had been examined in only two cases (Garnier, 1891, and Morax, 1922). Recently I added two more cases (first section). Experiments on contusion in animals have been carried out several times, but although they yielded interesting information they failed to reproduce traumatic glaucoma.

Consequently the present knowledge of this subject is limited. Theories as to the origin of this traumatic rise of tension cover the whole field of glaucoma, but their value is proportional to the number of cases (usually only a few) observed by the authors. Nevertheless, this value is enhanced by the small total number of observations recorded. For this reason they may be briefly reviewed here. The following opinions have been expressed from time to time:

1. Traumatic glaucoma, if it occurs at all, must be uncommonly rare (A. von Graefe, 1869).

2. Traumatic glaucoma does not exist; all cases representing those of predisposed patients (Lagrange, 1922).

According to other writers, glaucoma is caused by:

3. Dislocation of the lens (C. and H. Fromaget and Thiel). This cannot be the cause, as glaucoma may occur after the lens has been expelled (Morax).

4. Accumulation of fluid in the choroid through blocking of the perivascular lymphatics with loose cells (Garnier).

5. Tear of the ciliary muscle, interfering with filtration at the angle of the anterior chamber (Garnier and Stoewer).

6. Pushing of the iris into a rupture of the sclera, with closing of the angle of the anterior chamber (Fuchs).

7. Rise of the albumin content of the aqueous humor through vascular paralysis (Peters and Sala). This is impossible, as the rise of pressure precedes the rise of the albumin content (Leplat and Magitot).

8. Thrombosis of an artery in the ciliary body (Garnier).

9. Derangement of the vascular innervation (Garnier), causing edema of the ciliary body with displacement of the angle of the anterior chamber (Stoewer).

10. Irritation of a sympathetic nerve fiber, causing hypersecretion and obstruction of filtration through vasoconstriction (C. and H. Fromaget), or causing variation of the volume contents of the eye (Marx and Quartero).

11. Vasomotor reactions through influence of the sympathetic nervous system (Magitot, Leplat and Schmidt and de Decker).

12. "Passive hypertony" through obstruction of the oribital circulation caused by an orbital hematoma (Magitot). This theory is not convincing (see later comment).

13. Hemorrhage (Morax).

As there is no consensus on this point, the subject is well worth further study.

Experiments (Leplat, Schmidt and de Decker and others) have demonstrated that in animals contusion of the eyeball causes a short period of hypertony followed by a longer period of hypotony but that glaucoma does not supervene. The tension readjusted itself in a few days even after a powerful blow.

Cases reported by Leplat, Magitot and myself show that in man also contusion is followed by variations of the intra-ocular tension. It is difficult to say whether hypertony usually occurs immediately after contusion, to be followed later by hypotony, or whether hypotony occurs at once without hypertony. Obviously our data lack accuracy, as one dares not measure the tension more than once each day, while in animals one can take a reading every few minutes. Suffice it to state that in a number of cases after a simple contusion I found a rise of tension followed by a fall of tension but that in another series of cases I found only hypotony. In every case, however, the intra-ocular tension in the affected eye differed from that in the sound eye for a number of days, sometimes for weeks. In the end the tension returned to normal.

In a number of other cases glaucoma supervened. Why did the tension return to normal in some cases? Why not in every case? What was the difference between these two groups of cases? One must endeavor to deduce this difference from the anatomic and clinical evidence.

The difference between these two groups of cases, those of traumatic glaucoma and those of simple contusion, is obvious. Serious intra-ocular lesions associated with glaucoma were present in the one group and insignificant lesions with only hypertony or hypotony in the other. These lesions, therefore, must give the key to the problem.

Before discussing them, however, other possibilities must be viewed. It has been stated by Lagrange that traumatic glaucoma does not exist, all patients with glaucoma following trauma representing patients who are predisposed to an attack of glaucoma. It is, of course, impossible to exclude this possibility in all cases. Yet it is difficult to accept Lagrange's opinion if one recollects that in almost every case contusion is followed by variations of tension. It is also difficult to believe that traumatic glaucoma in childhood is due to predisposition, as other forms of glaucoma are excessively rare in this period of life. However, it is

well to keep this possibility in mind when it is necessary to operate on older patients and to perform iridectomy in preference to paracentesis of the anterior chamber.

Magitot (1917-1918) expressed the opinion that the globe, although normal, may suffer from passive hypertony through obstruction of the extrabulbar drainage channels by an orbital hematoma. However, the globe had been examined from two to five days after the accident. In every instance the globe may have been injured, as a slight intra-ocular hemorrhage may easily be absorbed in that period. The fact that in some cases hypertension and palpebral hematoma declined together may have been accidental. Furthermore, the presence of a palpebral hematoma combined with normal movements of the globe (1917, first and fourth cases) hardly proves that an orbital hematoma or edema is present. Moreover, in the fourth case, which has just been mentioned, hemorrhage into the iris was seen, which proves that the globe had been injured. In another case published in 1918 (first case), the patient was examined two days after contusion. Finally, in one case (1917, seventh case) in which there was a fairly large hematoma of the eyelids, hypotony was noted instead of hypertony. Therefore, the theory of passive hypertony lacks sufficient evidence, and all these cases must probably be classified with those of other authors in which there was active hypertony.

If one cannot accept predisposition or retrobulbar obstruction as a factor, glaucoma must be caused by the traumatic lesions which one observes in nearly every case. An intra-ocular hemorrhage may be a bar to normal filtration, as in cases of hyphemia after operations for cataract. A late hemorrhage also may exert a bad influence on the course of events, as is illustrated by my fourth case and by Cordes and Horner's cases. This cannot be a frequent cause, as many investigators noticed that glaucoma occurred after the blood had been resorbed (e. g., my sixth case). In my first case also the tension rose a long time after the second hemorrhage had been resorbed. Moreover, glaucoma may occur without hemorrhage.

It is unlikely that iridodialysis causes high tension, as in iridectomy for glaucoma a piece of the iris as broad as possible is removed. Nor can paralysis of the ciliary muscle through tearing be held responsible, as paralysis of the oculomotor nerve is not accompanied by glaucoma. The vascular lesions cannot be held responsible, because the collateral circulation of the eye is good and because vascular lesions alone do not greatly influence the circulation (Dietrich). Moreover, no thrombi were observed in the ciliary vessels and vortex veins.

Dislocation of the lens is often accompanied by hypertension, so that glaucoma in such cases is usually ascribed to dislocation of the

lens (Thiel, in Schieck and Brückner's "Kurzes Handbuch der Ophthalmologie"). It is true that glaucoma with traumatic dislocation of the lens as a rule has an unfavorable course. Hegner, for instance, found glaucoma in eighteen of twenty-two instances of traumatic subluxation of the lens collected from the clinic at Jena. In every case the result of treatment was unsatisfactory. Nevertheless, dislocation of the lens is not accompanied by glaucoma in all cases, as Hegner found only eighteen cases of glaucoma in a total of forty-eight cases of traumatic dislocation of the lens collected from the clinic at Breslau. Among eleven cases of complete dislocation this author found glaucoma in only two, while among fifteen cases of dislocation into the anterior chamber (in eight cases traumatic and in seven spontaneous) glaucoma occurred in fourteen. Consequently, glaucoma supervenes only in the majority of cases of dislocations into the anterior chamber of the eye.

It has already been pointed out that in the cases reported in the literature the percentage of dislocations of the lens is greater in cases in which the course is unfavorable, but also that the percentage of other serious intra-ocular lesions is higher. This fact has issued from a comparison of all the instances collected from the literature, and it suggests that dislocation of the lens must be regarded simply as a symptom of serious damage to the eye. This is well illustrated by the two globes the microscopic features of which I have described. Moreover, it is well known that congenital dislocation of the lens may exist quietly for a long time. The fact that glaucoma sometimes manifests itself in these cases cannot be applied to normal eyes. It is also known that a dislocated lens may lie in the vitreous for years without causing any disturbance. Dislocation of the lens alone, therefore, is no direct cause for glaucoma. This is best illustrated by cases of dislocatoin of the lens in which glaucoma occurs if the patient lies prone and in which the tension returns to normal as soon as the patient lies on his back (Beccaria, 1893; Sabata, 1931). Even dislocation of the lens into the anterior chamber without glaucoma has been described (König, 1895). Finally, traumatic glaucoma is seen so often without dislocation of the lens that one may safely conclude that dislocation of the lens (excepting anterior dislocation) plays no leading part in causing traumatic glaucoma. In one of my own cases (case 12) dislocation of the lens was even accompanied by hypotony.

Regarding the relation between cataract and traumatic glaucoma, it may suffice to state that in the vast majority of cases of traumatic glaucoma no cataract was noted.

Finally, lesions of the nervous system remain to be discussed.

Clinically, the influence of lesions of the nervous system is demonstrated by the occurrence of glaucoma with herpes zoster.

Experimentally, also, there is enough evidence to demonstrate this nervous influence. In experiments on contusion in rabbits Leplat and Schmidt and de Decker noticed that the tension of the sound eye varied with the tension of the other eye, although to a lesser degree. Leplat also noticed, following hypertony, a rise of the albumin-content of the aqueous in both the sound eye and the injured eye. Magitot found that after placing a weight on one eye the tension rises above normal in both eyes. In addition, axon reflexes mediated by the trigeminal nerve are known to exert a powerful influence on the intraocular pressure (Duke-Elder) while the sympathetic nervous system also plays an important part.

In traumatic glaucoma the presence of lesions in the nervous system was deduced from the presence of corneal anesthesia by Garnye. Their presence must also be surmised in cases of traumatic glaucoma without visible signs of injury (Tillema, seventh case; Magitot, 1917, second case; Brand, 1905). The patients in the latter cases may have been predisposed, but I consider this unlikely in my case. In this case glaucoma was followed by hypotony. This is a reason for thinking that it represents a transitional stage between simple contusion and traumatic glaucoma. It will be remembered that many of the patients with simple contusion exhibited no lesions, although instability of tension was present in every instance. In such cases taken at random predisposition is highly improbable.

If the variations of pressure after contusion are caused by neurovascular reactions (Leplat, Schmidt and de Becker and others), it is reasonable to suppose that traumatic glaucoma is also based on such reactions. However, while simple hypertony and hypotony may be regarded as the reaction of the normal mechanism to a blow, traumatic glaucoma must be looked on as a pathologic reaction. In the former the healthy neurovascular system readjusts itself; in the latter it is hampered by lesions in the nervous system such as I have demonstrated microscopically.

PROGNOSIS

Previously I have drawn attention to the fact that in cases in which the course is unfavorable more serious lesions of the inner part of the eye are present. From this one may conclude that the presence of such serious lesions suggests a doubtful prognosis. Especially, dislocation of the lens and hemorrhage into the vitreous have proved to be bad signs.

TREATMENT

The cases reported by Magitot and by myself prove that in cases of simple hypertony and hypotony the condition readjusts itself without any treatment. It is sufficient to apply a protective bandage. As the

real cause is uncertain and as the effect of drugs is not clear, even in animals (Leplat's experiments), it is better to refrain from interfering. As glaucoma may manifest itself after every contusion, it is inadvisable to use mydriatics.

Although it was difficult to determine the influence of atropine or scopolamine, it is worthy of note that in the cases reported by Marx and Quatero, Myers, Sala (second case) and Cantonnet the mydriatic was replaced by miotics as soon as the tension rose and that in those cases visual acuity remained unimpaired. It gives one food for thought, however, that in cases reported by Peters (second case), Scheffels, Jacobson (first case), Brand, Hirschberg and Cordes and Horner (first and third cases) and, probably, also in one of my own cases, atropine was administered when the tension was high and that in those cases visual acuity was reduced in every instance. In one of Fromaget's cases atropine had been discontinued eight days before the onset of glaucoma, and yet visual acuity was reduced 1/10. In this case rupture of the sclera was present, which indicates that the eye had suffered a severe contusion and that the disease would have had an unfavorable course in any case.

One may conclude, therefore, that in a number of cases the course has been favorable with the cautious administration of atropine. Yet it seems inadvisable to use a powerful mydriatic, for one would not think of prescribing a mydriatic for patients predisposed to glaucoma. Others also have warned against the use of mydriatics in cases of contusion (Wagenmann; Hardesty, 1931). Green observed a marked rise of tension after the administration of atropine, while Luedde observed a similar effect of eucatropine. Moreover, the indication for the administration of mydriatics is not clearly defined. Some authors use it to obtain quick resorption of a hyphemia or to prevent the onset of "traumatic iritis." Hyphemias are generally resorbed quickly and spontaneously, while the occurrence of so-called traumatic iritis without a perforating wound is exceptional. Others administer a mydriatic to counteract hypotony. In the literature I have found no mention of cases of hypotony in which the course was unfavorable. This, then, need not be an indication. It is much more important to prevent glaucoma, the course of which is always uncertain, than to cure hypotony.

It is also advisable to warn patients against exertion for one week, as Cordes and Horner have observed severe hemorrhage with serious damage to vision.

Experience has shown that in many cases an attack of acute glaucoma is favorably influenced by pilocarpine and physostigmine. If miotics do not suffice one might consider paracentesis of the anterior chamber, because many observers have had good results. In older

patients and in cases in which paracentesis does not give quick relief it is better to carry out iridectomy, for two reasons: first, because experience has shown that this operation is generally efficacious in nontraumatic acute glaucoma and, second, because it is impossible to exclude predisposition for glaucoma in elderly patients.

It is difficult to outline a definite course of treatment for the other forms in which traumatic glaucoma manifests itself. The instances collected from the literature cannot teach one much here, because the cases in which the result of treatment was unfavorable are also those cases in which injury was most serious. At most, one can advise to try the operations for glaucoma in turn.

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DARK ADAPTATION AS A CLINICAL TEST

FURTHER STUDIES

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The study of dark adaptation is a study of a phase of retinal function, and if one accepts the von Kries theory it is specifically an evaluation of rod efficiency. In a previous paper I reported the results of the tests of dark adaptation obtained in a study of 1,008 eyes [1] and in another paper I presented a chart on which such findings may be plotted to enable one to compare the results more easily.[2]

Almost simultaneously with the beginning of dark adaptation there is a regeneration of the visual purple. Either some failure of this regeneration or a disease of the deeper structure of the retina or choroid may be the principal cause of pathologic dark adaptation. That the retina is richly supplied with vitamin A was shown by Yudkin and his collaborators [3] in experiments on animals in which healthy retinal tissue was fed to rats deprived of vitamin A in the diet, with improvement of their symptoms. Wolff [4] and Moore,[5] in examining sections of liver tissue chemically by the antimony trichloride technic of Carr and Price, using a Lovibond tintometer, showed the liver to contain the main reserve of vitamin A in the body.

A review is given in this paper of a study of 240 eyes (120 patients were examined); 229 were free from actual retinal disease. Vision, with the few exceptions stated later, was normal or nearly so. I divided into three groups the 120 patients examined. Each of these groups showed factors which might affect the nature of the graph for dark adaptation.

Group 1 included patients already described in my previous paper [1] but examination of whom gave further confirmation of the previously

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From the Laboratory of Ophthalmology, the Wills Hospital.

1. Feldman, J. B.: Dark Adaptation as a Clinical Test, Arch. Ophth. **15**:1004 (June) 1936.

2. Feldman, J. B.: A Graph for Recording Results in Dark Adaptation, Am. J. Ophth. **19**:510, 1936.

3. Yudkin, A. M.; Kriss, M., and Smith, A. H.: Vitamin A Potency of Retinal Tissue, Am. J. Physiol. **97**:611, 1931.

4. Wolff, L. K.: On the Quantity of Vitamin A Present in the Human Liver, Lancet **2**:617, 1932.

5. Moore, T.: Vitamin A Reserves of the Human Liver in Health and Disease, Lancet **2**:669, 1932.

found data. This group also included patients who were tested with various colors for light adaptation previous to the dark adaptation test. Ordinarily, white light is used in adapting the eyes of every patient to light before the studies of dark adaptation are made. There were also in this group a few patients with poor vision due to such conditions as amblyopia and conical cornea; patients with choroiditis, glaucoma or retinal detachment, and several patients with miscellaneous diseases.

Group 2 was composed of patients with a disease of the blood or with a pathologic condition of the blood vessels (arteriosclerosis). Since it is through the circulation that nutriment is brought to retinal tissue, it was thought worthy of study to determine whether a pathologic condition of the blood or the blood vessels might aid in disclosing a common factor as a cause for the pathologic graph.

Group 3 consisted of patients with diseases which have been found to be related to vitamin A deficiency. The lack of vitamin A might affect the results of the dark adaptation study. This group included patients with avitaminosis A, metabolic disturbance, calculi of the genito-urinary tract, diabetes mellitus or disease of the liver or gallbladder and correlated diseases, and pregnant and sterile patients.

METHOD OF STUDY

All the patients were studied in the usual manner.[1] The cases were unselected. When possible, a study of the blood was carried out. I have noticed that the only significant parts of the routine complete blood count seemed to be the percentage of lymphocytes and the color index, as related to the dark adaptation study; therefore, in the tables which follow I have incorporated only these two findings. In many cases it was impossible for one reason or another to obtain even these findings for purposes of analysis. In cases in which only one eye showed a pathologic condition in dark adaptation this is designated by "pathologic ½." "Pathologic +" and "pathologic + +" signify varying degrees of marked pathologic change in the dark adaptation graph. Focal infection was eliminated as far as possible in all cases. All the patients examined were adults, except for 5 diabetic patients (from 6 to 16 years old).[6] That age, per se, does not affect dark adaptation has been noted previously.[1]

Preexposure of Eyes to Light Other than White.—In all cases in which studies of dark adaptation are to be made the patient must look at a pure white light for a definite period. This is called light adaptation. In my cases this period lasted from three to five minutes.

6. These were from the clinic for patients with diabetes of St. Christopher's Hospital for Children.

Krause [7] quoted Kuhne, Boll and Weiner as noting a qualitative alteration of the visual purple when it is exposed to light of different colors. Red light changes the visual purple to a brownish color; blue light, to rose, and yellow light, to yellow. Two patients who had been previously tested and whose eyes had been checked as normal were used in this experiment. The preexposure to light in each case was three minutes, and in each test a different color was placed before the patient. As in my routine studies, each sitting in the test of dark adaptation was thirty minutes, and readings were taken every three minutes. Preexposure to yellow and red light delayed dark adaptation in both patients greatly beyond the thirty minute period. Only the preexposure to blue light did not materially affect the results of the dark adaptation studies to a great extent.

A review of the cases studied which were obtainable to date are presented.

STUDIES ON GROUP 1

Amblyopia.—Visual defects of various degrees do not usually affect dark adaptation to a serious extent. Surprisingly, the patient with dark adaptation marked pathologic ½ and 45 per cent lymphocytes (table 1) showed a normal dark graph for the amblyopic eye and a pathologic dark adaptation graph for the eye with normal vision.

Conical Cornea.—In my last study of dark adaptation I did not have any patients with conical cornea, although I felt at that time that visual error did not affect the dark adaptation.[1] Two patients were examined in this study. The vision of one was 3/60 for both eyes, with a concomitant convergent squint of 30 degrees. The other patient had vision of 6/60 in one eye and 6/12 in the other. Both patients showed a high normal dark adaptation reading. No studies of the blood could be made.

Choroiditis.—The two cases the data for which are given in table 2 are of great interest. In the first case the choroiditis was confined only to the macula of each eye, and yet the dark adaptation graph was pathologic, despite the fact that the macula is concerned only with light adaptation. In the second case a small patch of choroiditis was noted, but the important fact was the positive diagnosis of glaucoma associated with the normal graph for dark adaptation. In a case of this sort one would expect to find a pathologic graph.

Glaucoma.—These studies are based on reports of cases given in a previous article.[1] However, reports of studies of the blood were not included in that publication. All the patients were glaucomatous and had pathologic dark adaptation of various degrees. The two cases in

7. Krause, A. C.: The Biochemistry of the Eye, Baltimore, The Johns Hopkins Press, 1934.

which the dark adaptation was marked pathologic ½ merit a brief review. A. L. R., aged 51, had a condition diagnosed as glaucoma of both eyes. The tension at time of examination was 26 mm. in the right eye and 20 mm. in the left. Dark adaptation was normal in the right eye and pathologic in the left. The other patient, E. G., had a condition diagnosed as chronic simple glaucoma of both eyes; the right eye was blind. However, the left eye showed normal dark adaptation at this

TABLE 1.—*Data for Patients with Amblyopia* *

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
2	Epilepsy in 1 case	Normal	X	X
1	Epilepsy	Pathologic ½	45	—1
1		Pathologic ½	X	X
1		Pathologic	52	—1

* In this and the following tables X means that this test could not be performed.

TABLE 2.—*Data for Patients with Choroiditis*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	Macular choroiditis	Pathologic	40	X
1	Small patch of tuberculous choroiditis; juvenile glaucoma; tension 50; field now normal, with tension 18	Normal	36	—1

TABLE 3.—*Data for Patients with Glaucoma*

Number of Cases	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
2	Pathologic ½	45; 39	1
4	Pathologic +	26 (average)	—1
5	Pathologic ++	49; 44; 36; 50; 35	—1
1	Pathologic	41	1

sitting, despite the fact that the tension was 30 mm. (Schiötz) at the time. A résumé of the cases is given in table 3.

Miscellaneous Diseases.—An interesting group of 8 cases fall under this classification. A patient with a provisional diagnosis of myxedema showed definite signs of glaucoma, with a tension of 31 mm. and a contracted field. This patient had a normal dark adaptation graph. Another patient who had a dislocated lens and tension of 35 mm. also showed a normal graph. The patient with keratoconus associated with opacities of the vitreous in the left eye and with vision of 6/60 showed a normal graph for that eye. Three patients were suspected of having angioneurotic edema. All these showed a pathologic graph for dark adaptation.

Retinal Detachment.—This is a common cause of faulty regeneration of the visual purple (pathologic dark adaptation). Krause [8] quoted Andogsky as stating that he observed no regeneration of the visual purple after detachment of the retina of a rabbit.

STUDIES ON GROUP 2

This group, as has been mentioned, consisted of patients with diseases of the blood or the blood vessels. It is unfortunate that this group did not contain more outstanding examples of pathologic changes and

TABLE 4.—*Data for Patients with Retinal Detachment*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	High myopia	Pathologic +	X	X
1	Vitreous opacity	Pathologic ++	42	1+

TABLE 5.—*Data for Patients with Miscellaneous Diseases*

Provisional Diagnosis	Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
Beginning myxedema	1	Ocular hypertension; tension 31 mm. [Schiötz]); contracted field	Normal	29	1
Dislocated lens of left eye	1	Tension 35 (Schiötz)	Normal	X	X
Hypertension (arterial)	1	Blood pressure 210/130	Pathologic ½	X	X
Keratoconus and vitreous opacities of left eye	1	Vision 6/6, 6/60	Normal	53	—1
Chronic arthritis	1	Three infected teeth; low grade ethmoiditis	Normal	37	1
Angioneurotic edema (?)	3	Eosinophils 4 per cent in each case	Pathologic	32; X; 44	—1; X; 1+

that, for instance, a patient with 9 per cent eosinophils should be regarded as showing evidence of disease. A comparison between this patient and the patient with trichinosis with 30 per cent eosinophils is enlightening. The latter patient had no other illnesses and was improving from his infestation. He had a normal dark adaptation graph. It would seem from this that it was not the eosinophilia but the underlying pathologic condition which really affected the results of the dark adaptation study. So also in the other blood diseases it appeared that when the condition was active the graph showed evidence of a moderately pathologic condition, whereas in the cases in which the patient was on the road to recovery the graph was normal or nearly so.

8. Krause,[7] p. 82.

Of 5 patients showing a rise in the percentage of eosinophils 1 had postoperative uveitis; vision in each eye was perception of movements of the hand. This patient had active involvement, with 15 per cent eosinophils. The dark adaptation graph showed evidence of marked pathologic changes.

There were 2 cases of chorioretinitis. One patient had 8 per cent eosinophils and dark adaptation that was classed as pathologic + +. The other had 9 per cent eosinophils; the disease was not as severe

TABLE 6.—*Data for Patients with Diseases of the Blood*

Diagnosis	Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
Secondary anemia...	2	.....	Pathologic ½	23; 14	1+; —1
Pernicious anemia...	2	.....	Pathologic	48; 22	1+; 1+
Pernicious anemia...	1	.....	Normal	22	1+
Secondary anemia...	1	.....	Normal	20	1+
Lymphatic leukemia	1	.....	Pathologic	95	1+
Hodgkin's disease and syphilis	1	Interstitial keratitis; Wassermann reaction 3+	Pathologic ½	51	—1

TABLE 7.—*Data for Patients with Arteriosclerosis*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
2	Hemorrhagic retinitis	Pathologic +	X	X
3	.....	Normal	X	X
1	.....	Normal	42	—1
1	.....	Pathologic ½	X	X
1	Hypertension	Pathologic	33	—1
1	Hypertension	Pathologic +	X	X
1	Hypertension	Pathologic	35	—1
1	.....	Pathologic +	28	—1
1	.....	Pathologic	38	1+
2	Chronic nephritis; secondary anemia	Pathologic	19; 42	—1, —1
1	Secondary anemia	Pathologic +	23	—1
1	Hypertension	Pathologic +	X	X

as in the first case, and the graph did not show evidence of as pathologic a condition. Further observations might well be made in subsequent cases on the presence of eosinophilia in association with chorioretinitis.

There was 1 patient with marked arteriosclerosis and hypertension who had 9 per cent eosinophils and dark adaptation that was classed as pathologic + +. This patient was 71 years of age.

Arteriosclerosis.—In a previous review [1] it was pointed out that arteriosclerosis gave pathologic dark adaptation readings for all but 4 of the 31 patients examined.

The group now reported on consisted of 16 additional patients. The results were somewhat similar to those in the former study, as 12 of these patients showed a pathologic graph.

STUDIES ON GROUP 3

This group consisted of patients with such diseases as are supposed to be associated with vitamin A deficiency.

Vitamin A is colorless, belongs to the alcohol group and has the formula $C_{20}H_{29}OH$ and an absorption band at 324 angstroms (the ultraviolet area). The liver contains the main reserve of vitamin A in the body; vitamin A is also found to a great extent in the retina.[9]

Mathews [10] quoted Wald as accounting for the presence of vitamin A in the retina as follows:

1. Vitamin A is extracted from the blood by the retina, more by the pigment layer than the rods, and combines with protein to make visual purple.

2. When visual purple is exposed to light it becomes visual yellow (a photochemical change). The visual yellow then breaks up (a chemical change) into vitamin A and colorless products. The process is then repeated, vitamin A combining with protein to form visual purple. What little vitamin A is lost in the process is replenished from the blood.

Tansley [11] has shown by photographing various retinas the quantitative difference in regeneration of the visual purple in normal rats and rats with vitamin A deficiency. It was previously noted that a delay in regeneration of the visual purple frequently caused dark adaptation to become pathologic.

Eusterman and Wilbur [12] claimed frequent causes of vitamin A deficiency to be: (*a*) the growing demand of the tissues of the body of the child; (*b*) hepatic and intestinal disease in adults with poor absorption and storage; (*c*) an increased demand for vitamins, as in disease or infections. Xeropthalmia is the most common sign of vitamin A deficiency, but it is not necessarily a constant evidence of the presence of the condition. Moore [5] cited the cases of 12 patients who at autopsy showed no reserve of vitamin A and yet during life presented no evidence of xeropthalmia. Can a person who has enough vitamin A in the system suffer from avitaminosis? Mathews [13] expressed the belief that this is possible. He stated: "If the vitamin, which is an alcohol, esterifies with phosphoric acid before it can become active, and

9. Foster, J.: Vitamins in Ophthalmology, Arch. Ophth. **15**:1160 (June) 1936.

10. Mathews, A. P.: Principles of Biochemistry, Baltimore, William Wood & Company, 1936, p. 412.

11. Tansley, Katharine: The Regeneration of Visual Purple: Its Relation to Dark Adaptation and Night Blindness, J. Physiol. **71**:442, 1931.

12. Eusterman, G. B., and Wilbur, D. L.: Chemical Features of Vitamin A Deficiency, J. A. M. A. **98**:2054 (June 11) 1932.

13. Mathews,[10] p. 414.

if a protein component is also necessary for its activity, it might be possible by the action of a suitable phosphatase, acting on such a union to produce the symptoms of avitaminosis even when Vitamin A was present as such in the tissues."

The data for the patients examined in group 3 are given in the following sections.

Atrophic Rhinitis.—Cody [14] claimed that vitamin A is necessary to the nutrition of nasal, tracheal and aural epithelium. He quoted Glassheib and Fleishman as giving the pros and cons relative to the etiologic importance of vitamin A in atrophic rhinitis. Only 1 case of this type

TABLE 8.—*Data for Patients with Avitaminosis A*

Number of Cases	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	Pathologic	37	X
1	Pathologic	X	X
1	Pathologic	33	X
1	Pathologic	37	—1

TABLE 9.—*Data for Patients with Metabolic Disturbances*

Number of Cases	Basal Metabolic Rate	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	+17	Cystic goiter	Pathologic ½	X	X
1	+16	Anemia	Pathologic ½	50	1+
1	+17	Cystic goiter	Pathologic	39	1
1	—37	Myxedema	Pathologic	30	—1
1	—11	Prostatitis	Pathologic	37	—1

was observed; there was pathologic ½ dark adaptation. No tests of the blood could be carried out.

Avitaminosis A.—There were 4 cases in which vitamin A deficiency was suspected. In all the dark adaptation graph was pathologic.

Disturbances of the Basal Metabolism.—Few patients with metabolic disturbances could be obtained for study. Of the 6 patients whose basal metabolic rate was determined, only 1 had a definitely abnormal rate. This was a patient with myxedema. The reading was —37. In this case the dark adaptation graph was pathologic. The other patients in this group showed little pathologic change in dark adaptation. It will be noted that 1 patient with a normal reading (—11) for the basal metabolism also showed a pathologic graph, but this graph was not as pathologic as that of the patient with myxedema. In this particular case

14. Cody, C. C.: Relation of Vitamin A, D, B and G to Otolaryngology, Arch. Otolaryng. **16**:664 (Nov.) 1932.

there was definite prostatitis, which might account for the pathologic reading.

Calculi of the Genito-Urinary System.— Wolbach and Howe,[15] Higgins [16] and many others have found in avitaminosis A in the rat metaplasia of the tissue of the urinary tract and pelvis of the kidney, which resembled to a great extent the finding for a child who died of avitaminosis. This case was reported by Wilson and DuBois.[17] In the rat, however, in addition to the metaplasia due to avitaminosis A, stones usually form in the bladder or the kidney. McCarrison and Ranganathan [18] noted the importance of the rôle of vitamin A in the formation of calculi by saying that the phosphorus and calcium balance will not entirely prevent the formation of calculi unless a normal quantity of vitamin A is present.

McCarrison [19] expressed the belief that the metaplasia of the tissue of the urinary tract caused by avitaminosis A may form a nidus around which a deposit of calculus may form. That avitaminosis A is not the sole cause of calculi has been contended by Bliss, Livermore and Prather,[20] who found that from 38 to 43 per cent of rats did not show calculi when on a diet deficient in vitamin A. Higgins [21] found by laboratory experiments that calculi dissolved in rats who were fed on a vitamin A diet.

All these experiments were performed on animals. The Council of Pharmacy and Chemistry of the American Medical Association [22] in a review of the subject of vitamins found "inadequate evidence to warrant the claim that ingestion of vitamin A will prevent calculi in man."

The association between vitamin A and the visual purple was noted previously in this paper. It was also stated that faulty regeneration

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15. Wolbach, S. B., and Howe, P. R.: Tissue Changes Following Deprivation of Fat-Soluble A Vitamin, J. Exper. Med **42**:773, 1925.

16. Higgins, C. C.: The Experimental Production of Urinary Calculi, Urol. & Cutan. Rev. **38**:33, 1934.

17. Wilson, J. R., and DuBois, R. O.: Report of a Fatal Case of Keratomalacia in an Infant with Postmortem Examination, Am. J. Dis. Child. **26**:431 (Nov.) 1923.

18. McCarrison, R., and Ranganathan, S.: Researches on Stones: XII, Indian J. M. Research **19**:55, 1931.

19. McCarrison, R.: The Causation of Stones in India, Brit. M. J. **1**:1009, 1931.

20. Bliss, A. R.; Livermore, G. R., and Prather, G. O.: The Relation of Vitamin A and D to Urinary Calculus Formation, J. Urol. **30**:639, 1933.

21. Higgins, C. C.: Production and Solution of Urinary Calculi, J. A. M. A. **104**:1296 (April 13) 1935.

22. The Status of Certain Questions Concerning Vitamins, report of the Council on Pharmacy and Chemistry, J. A. M. A. **106**:1733 (May 16) 1936.

of the visual purple is caused by avitaminosis A. The phenomena in this faulty regeneration are instrumental in producing pathologic dark adaptation. I next attempted to ascertain the possible relationship between dark adaptation, renal calculi and avitaminosis A. Dr. William Ezickson [23] permitted me to examine 25 of his patients with calculi of the renal tract in this study. In addition to these, 3 other patients were examined who were obtained from other sources. The diagnosis in all the cases in this group was verified by roentgen, cystoscopic and other examinations. Two patients of this group had just passed stones. They were found to show no abnormality on roentgen examination, but one showed pathologic dark adaptation, and the other, pathologic ½.

Six months after the roentgen study that showed no abnormal condition in the patient with the pathologic graph, a return of symptoms made advisable a second roentgen study. This study gave evidence of recurrence of stones in the kidney. Unfortunately, I was not able to repeat the dark adaptation study.

For the remaining 26 patients in this group of patients with calculi of the renal tract definite results were obtained with the dark adaptation test. Two patients showed a pathologic ½ curve for dark adaptation. Twenty-three showed a pathologic graph. In the 1 remaining case an erroneous diagnosis had been made. There was no pathologic condition of the renal tract. The graph was normal. The color index in this case was —1, and the lymphocyte count was 26 per cent.

Of the patients with calculi, 15 showed a lymphocyte count of over 35 per cent. In 18 cases the color index was over 1. Although every patient with calculi in this series showed a pathologic curve, no definite degree of disease was disclosed. The number, size and composition of the calculi bore no descernible relation to the dark adaptation graph. In addition to the patient for whom the erroneous diagnosis of renal disease had been made a number of normal patients with normal dark adaptation graphs were examined, who acted as controls for the patients with calculi.

Diabetes Mellitus.—With the exception of 1 adult, all the patients examined were children whose ages ranged between 6 and 16 years. The graphs obtained in these cases varied from normal to mildly pathologic. Eight patients in all were examined. It is possible that the vegetables, eggs and butter included in a well balanced diabetic diet would tend to limit evidences of avitaminosis A in diabetes.

Diseases of the Liver and Gallbladder and Correlated Diseases.—Krause [7] quoted Bass and Koyanagi as associating diseases of the liver

23. A complete report of these cases will be published by Dr. Ezickson in a paper in the near future.

with a deficiency in regeneration of the visual purple and night blindness. I was fortunate in being able to study 2 patients with syphilis of the liver. Ordinarily syphilis does not affect dark adaptation. It is interesting to note that Wolff [4] found a normal amount of vitamin A

TABLE 10.—*Data for Patients with Diabetes Mellitus*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	.....	Pathologic	45	—1
4	.....	Normal	X	X
1	Coma on day before dark adaptation test was carried out.....	Pathologic	X	X
1	.....	Pathologic	X	X
1	.....	Pathologic ½	X	X

TABLE 11.—*Data for Patients with Diseases of the Liver and Correlated Diseases*

Diagnosis	Number of Cases	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index	Icterus Index
Ulcerative colitis.....	1	Pathologic ½	35	—1	....
Chronic nephritis.....	1	Pathologic	38	X	15.0
Cholecystitis.....	1	Normal	X	X	....
Chronic hepatitis and duodenitis	1	Normal	36	X	8.8
Chronic cholangeitis and hepatitis	1	Pathologic	26	X	...
Cholelithiasis; chronic cholecystitis; syphilis	1	Mildly pathologic	30	X	8.8
Chronic hepatitis.....	1	Normal	35	X	8.8
Syphilis of liver.....	1	Normal	32	X	...
Syphilis of liver.....	1	Pathologic ++	X	X	Marked jaundice

TABLE 12.—*Data for Pregnant Patients*

Number of Cases	Results of Dark Adaptation Test	Comment
4	Normal	
1	Pathologic	High myopia
1	Pathologic ½	

TABLE 13.—*Data for Sterile Patients*

Number of Cases	Results of Dark Adaptation Test
4	Normal
1	Pathologic

in syphilitic livers. One of the 2 patients in my series showed a normal graph, which was not unexpected. The second patient, in whom the disease was complicated by marked jaundice, showed a pathologic graph. Jaundice has been known to affect the graph in this manner.

Four nonsyphilitic patients with hepatitis were also studied; of these, 2 had a pathologic graph, and 2 had normal readings.

Pregnancy.—That vitamin A, together with vitamin E, is a factor in fertility and the processes of reproduction is conceded, and was mentioned by Cobb.[24] The 1 patient the data for whom are given in table 12 who had pathologic dark adaptation had myopia requiring a —12.00 sphere. Some investigators of dark adaptation have given high myopia as a cause of pathologic dark adaptation. Of the 6 patients examined in my series, 4 showed normal readings. All the patients were about six months pregnant.

Sterility.—Wilson and DuBois [17] found in the patient with sterility whom they examined post mortem a change in the columnar epithelium in the uterine mucosa to the stratified keratinized type. This change would, it is believed, explain the cause of some cases of sterility in which avitaminosis A was a factor. Five patients were examined in my series. Only 1 of these showed a pathologic dark adaptation graph. It was later found that tubo-ovarian disease was the underlying cause for the sterility in this patient.

SUMMARY AND CONCLUSIONS

A review of the results of dark adaptation tests on 120 patients (240 eyes) is reported. A pathologic dark adaptation graph may be due either to failure of regeneration of the visual purple or to a disease of the deeper structure of the retina or choroid. The cases were arbitrarily divided into three groups, according to the factor which was considered as affecting the dark adaptation graph. The groups are:

1. The group of patients with various diseases of the eye.

2. The group of patients with diseases of the blood or blood vessels, which included patients with anemia, patients with eosinophilia and patients with arteriosclerosis.

3. That group of patents in whom avitaminosis A was considered a factor in one or another of several diseases, e. g., renal calculi and metabolic disturbances.

An attempt was made to note the frequency with which pathologic dark adaptation was associated with an increased percentage of lymphocytes in the blood as well as with an increased color index.

An evaluation of different-colored lights for preexposure of the eye (light adaptation) showed that white light is the best and that blue light is next best. Dark adaptation was delayed with preexposure to red or yellow light in the normal eye.

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24. Cobb, Percy: Light Sense, in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, chap. 19, p. 206.

In cases of group 1 the following were noted:

1. In patients with defective vision due to such conditions as amblyopia or conical cornea the tendency is toward a normal dark adaptation graph.

2. Pathologic graphs are often obtained in cases of choroiditis and a great majority of the cases of glaucoma, chorioretinitis and retinal detachment.

In the cases of group 2 the results of the studies seem to indicate the following:

1. The basic cause of the blood condition was a material factor in determining the dark adaptation graph.

2. Arteriosclerosis in a great majority of cases gave a pathologic graph.

In group 3 the following were noted:

1. The patients with avitaminosis A all showed a pathologic graph.

2. Patients with metabolic disturbances tended to have pathologic readings.

3. Patients with calculi of the genito-urinary tract showed definite pathologic dark adaptation.

4. In diabetes mellitus no consistent results were obtained.

5. In diseases of the liver or gallbladder and correlated diseases the results were inconclusive.

6. Syphilis itself does not affect the dark adaptation graph. However, in the presence of marked jaundice it appears as though this would not apply.

7. Pregnancy and sterility do not materially affect dark adaptation. Examination of the blood was possible in only 75 of the 120 cases studied. In 45 of these 75 cases there was both a pathologic dark adaptation graph and a lymphocyte count of over 35 per cent. Only 5 patients with a normal reading for dark adaptation had a lymphocyte count of over 35 per cent. Two of these had a color index of over 1.

The triad of (*a*) a high lymphocyte count, (*b*) a color index of over 1 and (*c*) a pathologic dark adaptation graph was definitely observed in those patients with renal calculi. Fifteen of the patients showed a lymphocyte count of over 44 per cent. Eighteen showed a color index of more than 1.

The many conflicting results obtained in regard to pathologic dark adaptation are of great interest, and the subject merits further study, particularly in view of the fact that faulty dark adaptation is synonymous

with hemeralopia (night blindness). Attention has been called [25] to the many accidents which occur in airplane and motor service in both military and civil life which may be attributable to pathologic dark adaptation. With the foregoing thoughts in mind I shall attempt further study in this subject.

Drs. H. M. Eberhard, William Ezickson, J. D. Paul and O. J. Toland permitted me to observe a few of the cases here reviewed.

25. Adams, Dorothy: Report of Committee on Physiology of Vision: II. Dark Adaptation, Medical Research Council, Special Report Series, no. 127, London, His Majesty's Stationery Office, 1929. Percival, A. S.: The Light Sense, Tr. Ophth. Soc. U. Kingdom **40**:311, 1920. Park, I. O.: Preliminary Observations on Vitamin A Deficiency as Shown by Studies with Visual Photometer, J. Oklahoma M. A. **28**:357, 1935.

SPASM OF THE CENTRAL RETINAL ARTERY IN RAYNAUD'S DISEASE

REPORT OF A CASE

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In reviewing the literature on Raynaud's disease, it is found that ocular complications occur infrequently and spasm of the central artery of the retina is exceedingly rare. In fact, Raynaud's disease itself is not common.

Raynaud's disease was first described as a distinct malady in 1862 by Maurice Raynaud.[1] In describing Raynaud's disease, DaCosta[2] said that it is usually regarded as a vasomotor neurosis, and is seen most frequently in children and young women but sometimes in men. The etiology is not known. Cecil and Kennedy[3] said that the disease passes through three stages: local syncope, local asphyxia and local gangrene. Local syncope is characterized by vasoconstriction of the affected parts which makes them pale and cold. The fingers and toes are the parts usually involved, but the disease may affect the ears, nose, lips, chin and nates. There is a feeling of deadness, usually accompanied by severe pain or paresthesia of the parts. This stage is followed by local asphyxia or cyanosis, of a toe or finger usually. Local asphyxia may persist for weeks or months before gangrene starts. The parts are usually symmetrically affected, and attacks of Raynaud's disease may recur several times without ending in gangrene. The disease is seldom fatal.

It seems possible for spasm of the retinal vessels to occur in Raynaud's disease, since hemiplegia has been reported in several cases of this disease, which was probably due to spasm of the cerebral vessels.

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1. Raynaud, Maurice: De l'asphyxie locale et de la gangrène symétrique des extrémités, Paris, Rignoux, 1862.

2. DaCosta, J. C.: Modern Surgery, General and Operative, ed. 9, Philadelphia, W. B. Saunders Company, 1925, p. 103.

3. Cecil, R. L., and Kennedy, Foster: Textbook of Medicine, Philadelphia, W. B. Saunders Company, 1930, p. 1427.

Foster Moore [4] examined a number of patients specifically for spasm of the retinal vessels, but in no case was there any evidence of its presence at the time of examination.

Shinkle,[5] in 1924, reported a case of Raynaud's disease of the feet in a woman aged 44 years. Her left eye suddenly became blind, though the pupil still reacted to light. Her eye was examined by Drs. F. H. Lamb and Louis G. Heyn. They found the retinal arteries uniformly shrunken. Under the influence of amyl nitrite the retinal arteries dilated, but they contracted again as soon as the effect of the drug wore off. The patient was given large doses of potassium iodide and hypodermic injections of sodium nitrite. She gradually improved, and vision slowly returned to the left eye, but it never became normal.

E. B. Dunphy,[6] in a thesis read before the American Ophthalmological Society in 1932, reviewed the literature on the ocular complications of Raynaud's disease and also reported a case in which there was bilateral spasm of the central retinal arteries. The patient was a white girl aged 9 years, for whom the diagnosis at first was apparently rheumatic fever with purpura. After several months of slow improvement, attacks of local syncope, asphyxia and gangrene of the finger-tips occurred symmetrically, and the toes on each foot became cold and painful. The diagnosis then was thought to be Raynaud's disease. Six months after the diagnosis of rheumatic fever the patient complained of poor vision in the right eye. The house officer examined her and reported a "definite red papule lateral to the disk, with the rest of the retina pale." Dr. Dunphy examined her six months later, when she could only count fingers at 6 feet (182.8 cm.) with the right eye. At this time definite pallor of the right disk was noted, and the retinal arteries were extremely small. The retina was normal in color, and the red spot in the macula had disappeared. In a few days the left eye suddenly became blind. Dr. Dunphy reported blanching of the whole retina, with a cherry-red spot in the macular region. The arteries were extremely thin. The patient was given inhalations of amyl nitrite; paracentesis of the cornea was done, and she was given spirit of glyceryl trinitrate, but with no effect on the visual acuity or the fundus picture. Later, bilateral lumbar sympathectomy, right cervical sympathectomy and removal of the stellate ganglion were performed. Two years later she could count fingers at 3 feet (91.4 cm.) with the right eye and had the barest perception of light in her left eye.

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4. Moore, R. F.: Medical Ophthalmology, ed. 2, Philadelphia, P. Blakiston's Son & Company, 1925, p. 95.

5. Shinkle, C. E.: Raynaud's Disease Involving the Feet, the Left Retina and the Heart Wall, J. A. M. A. **83**:355-356 (Aug. 2) 1924.

6. Dunphy, E. B.: Tr. Am. Ophth. Soc. **30**:420-430, 1932.

Allen and Brown [7] reported one hundred and forty-seven cases of uncomplicated Raynaud's disease at the Mayo Clinic during the period from January 1920 to July 1931. Of these patients, one had attacks of blindness suggestive of intermittent spasm of the retinal arteries.

Owing to the infrequency of spasm of the central retinal artery in Raynaud's disease, we should like to add the following case to those previously reported.

REPORT OF CASE

History.—K. G. E., a white man aged 39 years, was referred to us by Dr. O. W. Leonard on Oct. 23, 1936, because of sudden blindness of the right eye on October 10, which had persisted. There was no history of trauma or previous disease of the eye, and the blindness was not accompanied by pain. The family history offered nothing of interest other than that the patient was of German-Irish descent. The past history was interesting and of particular importance. The patient had had Raynaud's disease for eleven years, since 1925. He suffered with numbness and cold toes in 1925, accompanied by severe pain. This resulted in gangrene and loss of part of the great toe of the right foot. He had no further trouble until 1930, when he had a similar attack resulting in gangrene of the second and third toes of the right foot; these were amputated completely. He was a radio operator and went about his work with only an occasional attack of coldness, numbness and pain of the fingers and toes until 1935. At this time he had a similar attack, but more severe, which resulted in gangrene and the loss of the greater part of the second and third fingers of the right hand. He has had no gangrene of the affected parts since 1935 but frequently has recurrent attacks of cold fingers and toes, with numbness and pain. The condition has aggravated him to such an extent that he resorts to alcoholic stimulants at least twice a week and probably more often. He has smoked from twenty to thirty cigarets daily for the last twenty-two years.

Examination of the Eyes.—Vision of the right eye was limited to the barest perception of light in a very small area of the temporal field. Vision of the left eye was 20/20. The right fundus showed a pale, opaque, milky-white retina, especially in the region of the fovea centralis and the optic papilla. There was a bright red spot in the center of the fovea. The retinal arteries were very thin and small, and several branches were bloodless; the retinal veins were apparently normal. The arteries in the left fundus were slightly smaller than the average, but the veins appeared normal. The right fundus of this patient showed a typical textbook picture of embolism of the central artery.

Diagnosis.—There seemed to be no doubt as to the diagnosis of Raynaud's disease, since the patient had been seen by many physicians, who had concurred in this diagnosis. In view of this diagnosis and the history of the patient, our diagnosis was spasm of the central artery rather than an embolus. Broken columns of blood could be seen moving slowly in the arteries away from the disk, more rapidly at some times than at others.

Treatment and Course.—Since this eye had been blind for thirteen days, we realized that although the retinal circulation were restored the vision would not return because the retina would already be atrophic. However, we advised imme-

7. Allen, E. V., and Brown, G. E.: Raynaud's Disease: A Clinical Study of One Hundred and Forty-Seven Cases, J. A. M. A. **99**:1472-1477 (Oct. 29) 1932.

diate hospitalization for possible lumbar and cervical sympathetomy for the Raynaud disease and at the same time to see whether we could restore normal retinal circulation. The patient did not consent, and we did not see him again until December 4.

In the interim he was seen by Dr. E. W. Carpenter,[8] who will probably give a report of the end-result at a later date. When the patient returned to us on December 4, vision of the right eye was not improved. The retina was normal in color; the red spot in the fovea centralis had disappeared, and the arteries were somewhat larger. More blood was flowing through the arteries, but the smaller branches still appeared thin and bloodless. The disk showed definite pallor, which was more marked on the temporal half. The left fundus was essentially normal. The patient complained of the fact that his toes and fingers became cold and numb; so he was given large doses of potassium iodide. By December 29 the coldness and numbness had disappeared again.

When he was last seen, on Jan. 22, 1937, vision of the right eye was still only the barest perception of light in a small area of the temporal field. The fundus picture was essentially the same as on Dec. 4, 1936. He had not been bothered with coldness and numbness of his toes and fingers for over three weeks and was still taking potassium iodide.

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8. Carpenter, E. W.: Report by letter to the author.

THE GLAUCOMA CLINIC OF THE HERMAN KNAPP MEMORIAL EYE HOSPITAL

MARK J. SCHOENBERG, M.D.
AND
BENJAMIN ESTERMAN, M.D.
NEW YORK

For many years ophthalmologists have realized that progress toward the solution of the problem of glaucoma has been so dishearteningly slow that something more definite must be achieved in regard to this question.

The symposium on glaucoma at the Thirteenth International Congress of Ophthalmology, held in Amsterdam, the Netherlands, in 1929, did nothing to improve the situation. Duke-Elder, Magitot, Wessely and von Haagen, who reviewed the subject, confessed that the mystery was as profound as ever.

Having studied the problem for many years, our colleagues and we felt that the prevailing theories were leading nowhere. Gradually one of us developed a theory of his own as to the nature of glaucoma, differing in many respects from prevailing theories. We felt, also, that no progress would be made except by means of a carefully thought out plan of action not only involving theoretical considerations and laboratory investigations but combining these with a well organized, systematic and intensive clinical study of each patient with glaucoma.

With these ideas in mind a special clinic for patients with glaucoma was opened at the Herman Knapp Memorial Eye Hospital in October 1935. A modest sum of money was granted by the trustees of this institution. The staff of the clinic consisted of a director, an assistant and a medical diagnostician. The patients were referred to the clinic by the members of the staff of the Herman Knapp Memorial Eye Hospital. After a careful examination of the eyes each patient was referred to the medical diagnostician, who, in addition to recording the findings of the physical examination, recorded the personal and the family history as completely as possible. With all the available data at hand, the staff of the clinic was better able to see the entire picture and to be guided by it when prescribing treatment. Were space not limited, a number of instances could be cited in which the course of glaucoma was unmistakably influenced by constitutional or endocrinologic aberrations.

The examination of the eyes in each case was done in a leisurely fashion and thoroughly, at intervals varying from once a week to once a month. This, in addition to routine tests, included slit lamp study, tonometric examination and refraction. The peripheral and central fields were checked at least every six weeks. Special emphasis was laid on every detail of the patient's complaints. Nothing that the patient had to say regarding his ocular or general condition was considered insignificant. The fact was not lost sight of that in patients with glaucoma the threshold between the eye and the impulses reaching it from the rest of the organism is lowered; that a cold in the head, a febrile disease, a mental upset, a gastro-intestinal disturbance and other conditions often induce a rise of the ocular tension, sometimes of considerable proportion. Inquiry was made not only into the physical condition of the patient but also into his mental life. Lately, the addition to the staff of a psychiatrist for the sole purpose of studying the patients from the psychiatric point of view has been considered.

The clinic succeeded in securing the services of a full time ophthalmologist, intensely interested in the problem of glaucoma, for the routine examination of the eyes of each patient. This gave the other members of the staff more time to study the entire picture in each case and particularly any interesting or unusual features.

The examination of the ocular tension at regular intervals and at a definite time after the use of miotics furnished a formula as to how often drops had to be used in each case in order that the tension might be maintained within safe levels. Of course, a patient who needed too frequent instillations was sent back to the service from which he was originally referred, for operative procedures.

A glandular extract and a new drug were used in some cases. These have been employed for too short a time and in too small a series of cases to warrant any definite conclusions.

As the purpose of the clinic was not only diagnosis and treatment but also research work along clinical lines, a problem from among the many confronting the clinic was allotted to each of the members of the staff.

Almost at the beginning of the work it was found that most of the patients did not understand the nature of their ailment and the importance of full cooperation. Naturally, limitation of time in the average clinic makes detailed instruction in this direction impossible. Most of the patients do not know how to instil drops, nor are they aware that sitting for a few hours in the dark (as in motion picture theaters), becoming excited, neglecting chronic colds in the head, drinking much strong coffee, and other practices may counteract the salutary effect of miotics. A leaflet with instructions printed in large type was given to

each patient, and arrangements were made for the numerous extramedical problems to be handled by a well trained social worker. The National Society for the Prevention of Blindness cooperated in this matter. This society has agreed to supplement the salary of a social worker for the period of one year as a demonstration of what might be accomplished by social case work and follow up.

Almost from the outset the rapid accumulation of clinical data showed the necessity of systematization of records for the sake of ready reference and to make possible future statistical studies. After a number of revisions the final form adopted consisted of six record charts: Charts I and II are for the ocular and the medical history, respectively, and chart III, for the data obtained by examination of the eyes. Chart IV is for a systematic record of the subsequent course, which is checked on each revisit. On chart V is registered the social factors which may have a bearing on the disease, and chart VI contains a summary of the entire clinical picture and an outline of the projected course of treatment.

These charts have been kept as concise as was practicably possible and in actual use have been found to be the means of saving a great deal of time, once one has become familiar with them.[1] The two most useful (charts I and IV) are reproduced here, showing the history and the follow up in a typical case.

In chart I (fig. 1) the data in the first four columns in section E indicate that in 1928 the patient began to experience slight (1 +) fogging of vision and halos in the left eye, and in 1930 there were marked (4 +) loss of vision, ocular pain and headache with nausea and vomiting; that in November 1934 she noticed slight (1 +) fogging of vision, pain in the supra-orbital region and halos in the right eye, and that in July 1935 she became aware of diminished visual acuity and frequently stumbled over objects of furniture.

The next two columns (F and G) give more information about the aforementioned symptoms, correlated with the numbered lists of aggravating and relieving factors. It is seen that the patient's symptoms were aggravated at certain times of the day (column F, 1), when she was worried (column F, 4) and during colds in the head (column F, 6), but were relieved by rest (column G, 22) and hot applications (column G, 26).

The two columns to the left of column F are for details of the first and last acute attacks. In this case there was only one attack, in the left eye, in 1930, lasting seventy-two hours, brought on by "trouble

1. The space is too limited to allow the publication of the records and explanatory notes. We shall be glad to mail an exemplar of each to those desiring to obtain them.

at home" (column F, 5) and relieved only by enucleation (column G, 31). The attack consisted of 4 + loss of vision, 4 + ocular pain, 4 + headache and 2 + nausea and vomiting.

Section H indicates that the glaucoma was discovered in 1930 (the time of the patient's acute attack in another clinic and that she was given drops for the right eye and advised to have an operation on the left eye. It is also learned that she had been to an optometrist in 1928 and 1929, and a glance back at column E will recall that the fogging of vision and the halos in the left eye started in 1928. "(Over)" refers to a space on the reverse of the chart, left blank for the addition of

GLAUCOMA RECORD – EYE HISTORY I

CLINIC NO 209412

A. NAME a R | AGE 51 | SEX F | M (W) S D SEP. DES. | CLINIC Knapp | SERIAL NO

ADDRESS 642 a Ave, Bx. | OCCUP. Chambermaid | RACE Wh | SERVICE OF Dr. Knapp | DATE OF HISTORY Nov. 12, 1935

B. DIAGNOSES 1. EYE — PRINCIPAL o.d. Chr. Simple Glaucoma; o.s. Anopl. Headache — SECONDARY o.d. Hyp. Astig., Presby.; o.s. — 2. GENERAL Hyperthyroidism, Sec. Anemia

C. OPERATIONS AND DATES: o.d. —; o.s. Iridectomy (?) 1930, Enucleation 1930

D. CHIEF COMPLAINT: O.D. Vision poor, 4 mo. Headaches and foggy vision for past year.

E. GLAUCOMA SYMPTOMS	SEVERITY 1+ to 4+ R	SEVERITY 1+ to 4+ L	DATE OF ONSET R	DATE OF ONSET L	BROUGHT ON BY SEE F INDICATE NO.	RELIEVED BY SEE G INDICATE NO.
FAILING VISION	3+	4+	7/35	'30	1, 4, 6	22, 26
FOGGING OF VISION	1+	1+	11/34	'28	1, 4, 6	22, 26
PAIN – OCULAR		4+		'30		
– SUPRAORBITAL	1+		11/34			
– HEADACHE		4+		'30		
HALOS (RAINBOW)	1+	1+	11/34	'28	1, 4, 6	22, 26
REDNESS						
NAUSEA, VOMITING		2+		'30		
CONSCIOUS OF FIELD LOSS	3+		7/35			
WEAK ACCOMMODATION						

1ST ACUTE ATTACK R◄L	LAST ATTACK R◄L
DATE 1930	DATE
DURATION 72 HRS.	DURATION HRS.
CAUSE F: 5	CAUSE F:
RELIEF G: 31	RELIEF G:

F. SYMPTOMS BROUGHT ON BY	G. SYMPTOMS RELIEVED BY
1. TIME OF DAY	21. SLEEP
2. FATIGUE	22. REST
3. USE OF EYES	23. SEDATIVE
4. WORRY	24. EYE DROPS
5. TROUBLE AT HOME	25. COLD APPLIC.
6. HEAD COLD	26. HOT APPLIC.
7. ILLNESS	27. CHANGE OF ENVIRONMENT
8. CONSTIP. OR INDIGESTIVE UPSET	28. HOSPITALIZED
9. MENSES	29. OPERATION
10. WEATHER	30. SPONTANEOUS
11. NO KNOWN CAUSE	31. enucleation
12.	32.
13.	33.

H. GLAUCOMA DISCOVERED
DATE 1930
BY WHOM other clinic
ADVISED: drops O.D. operation O.S.
DATE SEEN BEFORE GLAUCOMA DISCOVERED 1928, 1929
BY WHOM optometr.
DROPS STARTED (DATE) R: 1930 (over) L: —
AT PRESENT USING MO DROPS
FREQUENCY R: L:
TOTAL # ACUTE ATTACKS R: 0 L: 1

USE REVERSE FOR ADDITIONAL DATA AND DETAILS

SIGNATURE B.E. M.D.

Fig. 1.—Chart for the optical history.

notes, comments and sketches. Here it is stated: "Patient spontaneously discontinued drops and clinic visits in 1931 because she felt well"—an all too frequent occurrence in the absence of social service. On admission to the clinic of the Herman Knapp Memorial Eye Hospital the patient had been using no drops.

Having read all this, one can readily appreciate the value of being able to record so detailed a history on two thirds of an ordinary 8 by 5 inch (20.3 by 12.7 cm.) clinic card.

Chart IV (fig. 2) is almost self-explanatory. One line is used for each revisit and tells the facts noted as a routine each time. Any notes outside these may be added on the reverse side of the chart, left blank

for such purposes. Column 35 may be used for writing in an appropriate heading to keep a record of some special measure indicated and being tried in a case. Column 39 calls for a check or an initial each time that perimetric examination is carried out, making for greater regularity in field taking.

A good deal of effort was also spent in preparing a blank for recording the medical and psychologic abnormalities of the patient and, when possible, of the members of their families also. Dr. A. Baumann, the medical diagnostician of the glaucoma clinic, has worked out a record which we deem to be as complete as possible and helpful in cor-

NAME a R — GLAUCOMA — SUBSEQUENT RECORD IV — CLINIC No 209412 — SERIAL No

30 DATE	31. VISION WITH BEST CORR. R	L	32. MEDICATION R MED.	FREQ.	L MED.	FREQ.	33. HOURS SINCE LAST MED. R	L	34. TENSION (TIME) R	L	35.	36. Rx	37. REMARKS	38. EXAMINER	39. PERIMETRY
11-19-35	20/40	–	#6 4 x d		–		4	–	28	–		#6 5 x d	No fogging or halos	E	E
11-26	20/40+2	–	#6 5 x d		–		4	–	23	–		"	Basal metab: +15	E	
12-3	20/40+2	–	#6 5 x d		–		4	–	23			"	Rx: her usual gl.	E	
12-10	20/40+2	–	"		–		4		23			"		E	
12-17	20/40-3		"				4		23			" (buffered)	Redness & tearing 2d. Conjunctivitis	E	
12-31	20/40+1		" (buffered)				3		21			"	Better since using buffered #6	E	B
1936 1-14	20/40+3		"				3½		21			"		E	
1-28	20/40+2		"				20!		36!			"	Ran out of drops	E	
2-11	20/40		"				3½		21			"		E	B
2-25	20/40+2		"				4		23			"		E	
3-17	20/30-2		"				4		30			#6 q 2 h. Bromides	Trouble at home (see over)	E	
3-24	20/40-1		#6 q 2 h. Bromides				4		23			"		E	
4-7	20/40		"				4		23			"		E	B
4-28	20/40+1		"				3½		20			#6 4 x d. Disc. Bromides	New living quarters	E	
5-19	20/40		#6 4 x d				4		20			"	Better	E	

Fig. 2.—Chart for the subsequent history.

relating the ocular findings with the general physical status. A number of interesting and new observations were thus made and will be ready for publication when the number of patients is larger.

In the course of the work of the clinic the staff was confronted by a number of questions and difficulties which could not be answered or overcome without the cooperation of all the other ophthalmic institutions of New York.

The first question was: What are the distant results with the present day methods of examination and treatment of patients with glaucoma? Then, in what percentage of cases of glaucoma have ophthalmologists

succeeded in conserving useful vision during the past ten years? Does their work on glaucoma show better visual results during the past ten years than it did during the preceding decade?

Some who have been practicing ophthalmology for several decades doubt whether the results in glaucoma in the period from 1925 to 1935 are superior to those in 1915 to 1925. Whether or not this is so cannot be ascertained unless a thorough survey is made of the entire population with glaucoma in the dispensaries of New York. Such a survey is unquestionably of fundamental importance and must, of course, be made with all the care and accuracy necessary to secure reliable statistical data. Nowadays, the study of any problem—be it scientific, sociologic, commercial, etc.—is invariably begun by a careful appraisal of all the factors involved. A survey has the advantages of pointing out weak points or trends that lead nowhere or in wrong directions, faulty methods and other weaknesses.

The next question that arose was: Judging from our year's experience in the glaucoma clinic, do we feel that the patients are better handled, their diagnoses more completely worked out and the indications for various types of treatment more accurately established in a separate department than in the old-fashioned general clinic? The following facts are overwhelmingly in favor of special glaucoma clinics:

1. Among all the patients with ocular diseases, patients with glaucoma deserve to be placed in a special group. Their examinations are much more time consuming; each patient has to be carefully followed and studied. For this reason these patients cannot be treated in a routine fashion.

2. The psychologic approach to cases of this condition, in which mental upsets play an important rôle, must be performed with the same care and tenderness used in any surgical operation. In clinics for patients with thyroid disease a great deal of attention is given to this principle.

3. The social worker's function of disentangling complicated social and psychologic situations can be performed only in quiet and relaxing surroundings—not in the midst of the bustle of a general clinic.

4. Having to deal with a large number of patients with glaucoma, the ophthalmologists in the special clinic are given a splendid chance to acquire a vast amount of knowledge concerning the various phases of the problem of glaucoma, and those among them who have a bent toward research have a good opportunity to discover points previously unknown or to clear up questions previously misunderstood.

5. The patients quickly realize that special efforts are being made to help them, and they respond in a gratifying manner by cooperating to the full extent of their ability.

6. Year by year the carefully kept records grow in number and content and in the course of time furnish valuable material for study.

For these reasons, among others, we thought it advisable to call the attention of some of the ophthalmologists of New York to the advantages and possibilities of special clinics for patients with glaucoma. The response of our confrères has been prompt and favorable. Within a short time two colleagues have signified their intention to open special glaucoma clinics in other institutions, and there is no doubt in our minds that in due time others will join the ranks.

The idea of special clinics is not an entirely new one in medicine. There are already numerous clinics for persons with cardiovascular disease, diabetes, cancer, arthritis and other diseases. Many of those who are attending such clinics have formed local or national associations for study and exchange of ideas regarding the diseases of their special field.

Ultraspecialization in medicine is deprecated nowadays; the modern trend is toward correlation of all the findings furnished by a complete examination of the whole patient and, whenever desirable, of the whole family. By the same token patients with glaucoma should no longer be treated as though the only ailment was a pair of sick eyes for which but one of two things may be done: operation or the administration of miotics. Glaucoma clinics are meant to do for their patients what clinics for diabetes or clinics for cardiovascular disease are doing for theirs. They are meant to evaluate the entire physical, mental and inherited make-up of the patient, reconstruct a complete picture of the problem and try to do something for the whole patient instead of for his eyes alone.

Finally, not only the patient but the ophthalmologist can profit considerably from the special clinic for patients with glaucoma. The opportunity to see and follow such a group of patients will result in rounding out a knowledge of glaucoma which could hardly be considered complete even among the best ophthalmologists.

SUMMARY

Our one year's experience with the glaucoma clinic of the Herman Knapp Memorial Eye Hospital has taught us that such a clinic is fulfilling an urgent need, that it is in step with the spirit of modern clinical medicine, that it is bound to serve the best interest of the patients and of the community by preventing blindness whenever possible and that it offers opportunities for research unequaled by the average ophthalmic clinic.

It is our firm belief that progress is possible, if the problem of glaucoma is attacked by:

(*a*) A systematic and intensive medical as well as ophthalmologic study of each patient with glaucoma.

(*b*) Making a thorough survey of the glaucoma material in the ophthalmic institutions of the metropolitan area.

(*c*) Establishing a glaucoma clinic in each ophthalmic institution.

(*d*) Organizing an association of glaucoma clinics.

667 Madison Avenue.

MODIFIED SUBCONJUNCTIVAL EXTRACTION OF CATARACT

A PRELIMINARY REPORT

H. C. ERNSTING, M.D.
CLEVELAND

Subconjunctival extraction of the lens is not a recently developed procedure. It was recommended and performed early in the nineteenth century and has been more or less continuously practiced since that time.

Many modifications of the original technic have been developed, and only recently W. Moehle [1] described a method of subconjunctival extraction of the lens. It is evident, from the literature, that a progressive attempt has been made to modify and improve the technic of this type of operation.

Since its origin this method has been recommended for those cases in which loss of vitreous is threatened either during or after extraction of the lens.

The procedure which I am presenting is a modification of subconjunctival extraction which retains the qualities for which the extraction has been recommended and at the same time permits ease and simplicity of operation.

Before operation a complete history is taken, and the patient is given a physical examination. The laboratory examination varies in extent with the condition of the patient, but examination of smears from the eye, urinalysis, determination of the blood chemistry and a Wassermann test are carried out as a routine in each case. Each eye is irrigated with a 1:8,000 aqueous solution of merthiolate three times daily, and a 1 per cent solution of atropine is instilled into the cataractous eye the night preceding the operation and again on the morning of the operation. Anesthesia is produced by six instillations, given at intervals of three minutes, of a 4 per cent solution of either cocaine or metycaine. Sedatives are given preoperatively. A binocular dressing of mercury bichloride is applied before the patient is taken to the operating room.

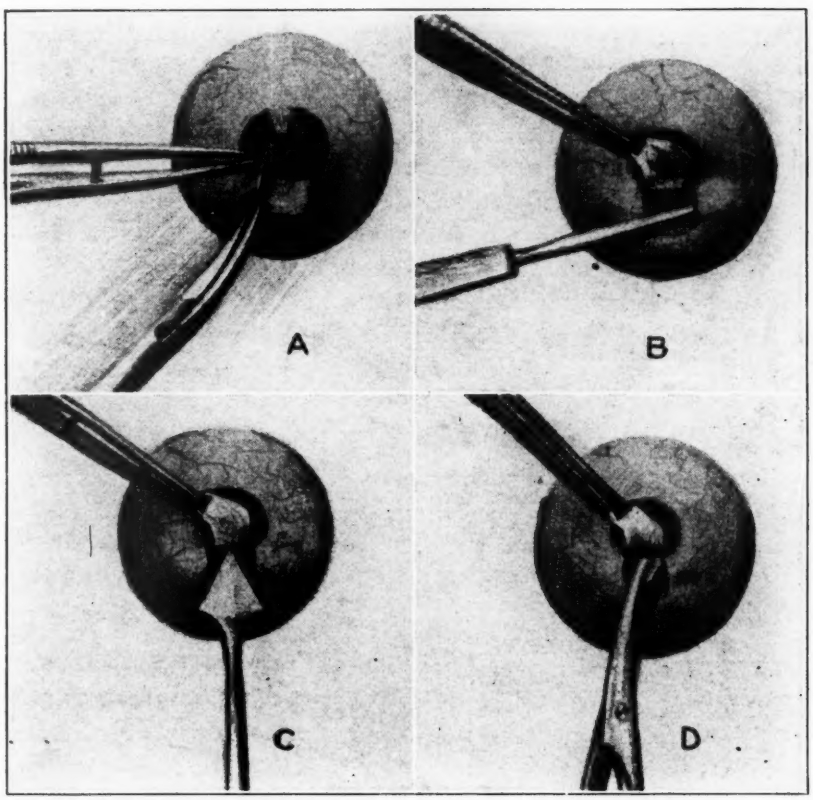
From the service of Drs. W. E. Bruner, P. G. Moore, G. L. Miller and B. J. Wolpaw.

From the Department of Ophthalmology of the Western Reserve University School of Medicine and the Cleveland City Hospital.

1. Moehle, W.: Cataract Extraction Through a Vertical Conjunctival Slit, Arch. Ophth. **16**:659 (Oct.) 1936.

TECHNIC OF OPERATION

The field of operation is cleaned and draped in the usual manner, and akinesis is produced by means of the method of O'Brien [2] or that of Van Lint.[3] The lid retractor is then inserted; the eye is irrigated with a 4 per cent solution of boric acid, and the patient is requested to look toward his feet so that adequate exposure of the superior part of the eye may be obtained.



A, a conjunctival flap approximately 6 by 4 mm. is made at 12 o'clock. This is dissected free from the underlying tissues up to the limbus. *B*, the conjunctiva on each side of the flap is undermined up to the limbus to 3 and 9 o'clock. *C*, a keratome is placed at the limbus, beneath the conjunctival flap, at 12 o'clock and is inserted into the anterior chamber in the usual manner. *D*, the primary incision is enlarged on each side to the extent of the undermined conjunctiva by blunt-pointed scissors, one blade of which is placed in the anterior chamber just anterior to the iris and the other blade of which is placed outside but beneath the conjunctiva which has previously been undermined.

2. O'Brien, C. S.: Local Anesthesia, Arch. Ophth. **12**:240 (Aug.) 1934.
3. Van Lint: Ann. d'ocul. **151**:420, 1914; Arch. d'opht. **43**:714, 1926.

A conjunctival flap approximately 6 by 4 mm. is then made at 12 o'clock. This is dissected free from the underlying tissues up to the limbus and is laid over the cornea to keep it out of the way.

The conjunctiva on each side of the flap is undermined up to the limbus to 3 and 9 o'clock by means of an iris repositor.

A keratome is then placed beneath the conjunctival flap at 12 o'clock and inserted into the anterior chamber in the usual manner. The primary incision is enlarged on each side to the extent of the undermined conjunctiva by blunt-pointed scissors, one blade of which is placed in the anterior chamber just anterior to the iris and the other blade of which is placed outside but beneath the conjunctiva which has previously been undermined.

A peripheral or basal iridectomy is then done, and the lens is extracted. The anterior chamber may be irrigated, if necessary.

The pillars of the iris are reposited, if necessary, and the conjunctival flap is replaced and smoothed over with a spatula. No sutures are required.

White's ointment [4] and a 1 per cent solution of atropine are instilled into the conjunctival sac. The lids are gently closed after it is ascertained that the flap is not displaced, and a binocular dressing is applied.

COMMENT

I have presented this modification of the subconjunctival type of extraction of the lens because it permits adequate working space and ease and simplicity of performance, while at the same time it retains all the qualities for which this type of extraction of cataract is recommended.

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4. White's ointment is prepared as follows: Mercury bichloride, 1:3,000, sodium chloride, 5:3,000, and an ophthalmic ointment base, to saturation.

PROGNOSIS OF POSTOPERATIVE SYMPATHETIC OPHTHALMIA

A STATISTICAL STUDY

HAROLD H. JOY, M.D.
SYRACUSE, N. Y.

There are many factors which make a study of collected cases of sympathetic ophthalmia difficult and somewhat unsatisfactory. The question of diagnosis is disturbing, as much depends on interpretation not only of the clinical signs but of the histologic picture. Unfortunately, histologic examination is so often omitted that the number of cases that can be collected in which the diagnosis has been confirmed is materially reduced. Then there are the questions of the type of therapy used and the time it was started, and the condition of the sympathizing eye when treatment was instituted. Furthermore, each case is an entity in itself, and the disorder may be dependent on entirely different factors from those in a similar disorder produced under the same conditions. This variation in the different factors which enter into sympathetic inflammation is confusing and unless carefully considered may lead to incorrect conclusions. Nevertheless, such a study may be of some value either in confirming the conclusions of other investigators or in demonstrating the possibility that no conclusions are possible in such a complex condition. In any event, it brings to the consciousness of ophthalmologic surgeons a hazard that is always present.

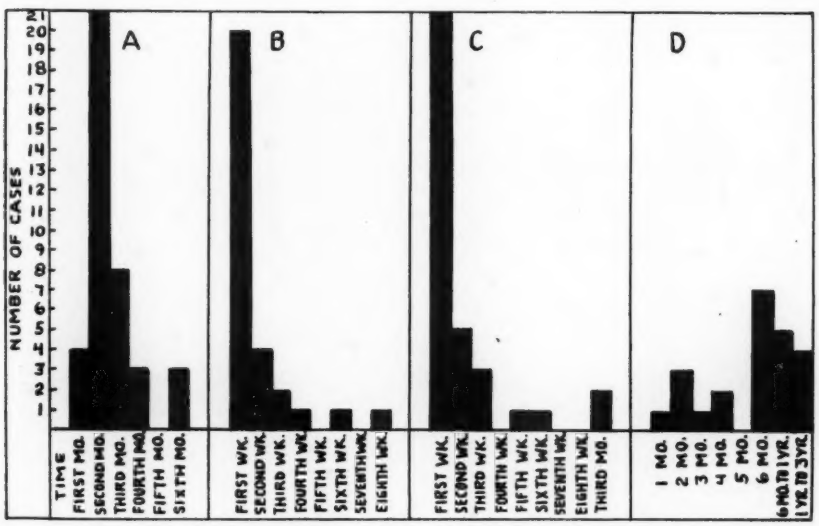
This study is based on one hundred and three cases of postoperative sympathetic ophthalmia collected by colleagues and from the literature. Chief consideration is given those cases in which the diagnosis was confirmed by microscopic examination. Of forty-four patients in whom an enucleated eye or a specimen of iris disclosed the characteristic histologic picture, one never showed sympathetic inflammation. Of the cases in which the diagnosis was proved in which the fellow eye later became involved, in twenty-six the disorder was attributed to operations for cataract, in sixteen it followed operation on the iris and in one it resulted from excision of the anterior segment of the globe.

Some other extenuating exciting factor was reported for twenty-five of the patients, in five of whom more than one such complication occurred. A definite preoperative pathologic process was observed in fifteen patients; an accident occurred during the operation or shortly

afterward in thirteen, and a second operation had been performed on ten. This phase has been discussed in another paper.[1]

The interval between the operation and the onset of sympathetic inflammation is shown in section *A* of the chart. It ranged from a minimum of two weeks in two cases to a maximum of six months, with by far the greatest incidence (84.6 per cent) within the first three months, particularly during the second month (53.9 per cent).

The exciting eye was removed in thirty-nine patients, for six of whom the time of enucleation is not recorded. In three instances sympathetic ophthalmia developed in spite of prophylactic enucleation, while in the remaining thirty patients the exciting eye was removed as a



Diagrams showing relationships in time in the series studied. *A* shows the interval between operation and the onset of sympathetic ophthalmia; *B*, the interval between the onset and enucleation; *C*, the interval between the onset and the beginning of treatment, and *D*, the duration of the condition.

therapeutic measure within three months after the onset of sympathetic inflammation, and in twenty (66.7 per cent) it was removed within the first week after the onset (section *B* of the chart). In five patients the exciting eye was not enucleated, but in three of these the inflammation in the sympathizing eye was so severe that it had to be removed.

Systemic therapy and treatment of the sympathizing eye were instituted within three months after the first signs of inflammation for all the thirty-five patients whose records are complete, and for twenty-six (74.3 per cent) they were begun within the first two weeks after the onset (section *C* of the chart).

1. Joy, H. H.: New York State J. Med. **36**:1498 (Oct. 15) 1936.

The condition in the sympathizing eye appeared mainly as iridocyclitis, except in three cases, in which it involved predominantly the posterior part of the uvea. The course of the disease was designated in twenty-six instances; it was reported as severe in fourteen patients, moderate in eight and mild in four. The duration ranged from one month to three years (section *D* of the chart), and, as would be expected, it was six months or more in the majority of cases (69.5 per cent).

Table 1 shows the relationship between the time at which therapy was begun and the outcome. In the few instances in which there was variation between the time of enucleation and that of starting other treatment the time of the first intervention is used to indicate the time of the beginning of therapy. All other factors being disregarded, this table shows a much greater proportion of good results in those patients

TABLE 1.—*Relationship Between Outcome and Time After Onset That Therapy Was Instituted*

Time After Onset That Therapy Was Instituted	Number of Cases	Outcome	
		Successful	Unsuccessful
Within 2 days	4	3	1
From 3 days to 1 week	17	12	5
Second week	5	4	1
First month	4	1	3
Second month	2	2	0
Third month	2	0	2
Definite interval not stated; within 3 months	2	1	1
Questionable	7	?	7
Total number of cases	43	23	20

who received prompt treatment. Thus, useful vision resulted in 73 per cent of the patients in whom treatment was started within two weeks after the onset, compared with 37.5 per cent of those in whom it was delayed one month or more. Altogether, of thirty-eight patients whose final vision was recorded, the outcome was satisfactory in twenty-one (55.2 per cent); in thirteen instances vision was 20/40 or better (table 2). Of the seventeen patients in whom treatment gave unsuccessful results, one retained vision of 1/25, while in the others it was reduced to light perception or total blindness.

Various authors [2] emphatically have maintained that sympathetic ophthalmia following extraction of cataract is especially severe and the outcome unfavorable. This was found to be true in the five cases in which the diagnosis was confirmed, reported by me,[3] and in one of

2. (*a*) Hambresin, M. L.: Bull. Soc. belge d'opht., no. 57, Nov. 25, 1928, p. 107. (*b*) Davids, H.: Arch. f. Augenh. **105**:13 (Oct.) 1931. (*c*) Jaeger, E.: Klin. Monatsbl. f. Augenh. **73**:714, 1924.

3. Joy, H. H.: A Survey of Cases of Sympathetic Ophthalmia Occurring in New York State, Arch. Ophth. **14**:733 (Nov.) 1935.

Woods' [4] two cases. Also, in E. Fuchs' [5] cases the condition was severe in four and moderately severe in one. On the other hand, Verhoeff [6] stated the belief that there are too many variable factors involved in this disease to enable one to arrive at a decision. Of the twenty-six cases in this series in which sympathetic ophthalmia resulted from operations for cataract, in nineteen the condition followed combined extraction of cataract of the senile type (table 3). All the patients but two were over 50 years of age. The incubation period ranged from four weeks to six months, and the duration from three to eighteen months. The condition in the sympathizing eye appeared mainly as iridocyclitis except in one case, in which it involved predominantly the posterior part of the uvea. In six patients in whom the exciting eye was enucleated and treatment was instituted within twelve days after the onset of sympathetic inflammation the outcome was uniformly good, all the patients attaining useful vision, while of three patients in whom the intervention was delayed from nineteen days to two months after the onset,

TABLE 2.—*Final Vision*

Vision	Number of Cases	Percentage	
20/ 40 - 20/15	13	34.2	55.2
20/200 - 20/50	8	21.0	
Perception of light - perception of fingers	5	13.2	44.8
No perception of light	12	31.6	

only one showed successful results. The exciting eye was removed in two patients two weeks and one month, respectively, before any signs of inflammation were observed in the fellow eye, but blindness resulted in both after a moderately severe course. The exciting eye was not removed in four patients (table 4). In one, in whom the diagnosis was made from a specimen of iris, both eyes escaped with useful vision, despite a delay of forty-two days in starting treatment. In the other three patients the sympathizing eye was so severely involved that it had to be removed. This also occurred in three of the cases in which the diagnosis was unconfirmed. Jaeger,[2c] in reporting thirteen of these rare cases, gathered from the literature and from the Tubingen clinic, found that in all except two the condition was due to extraction of cataract. In all three of the cases of this nature reported in the present series the condition followed multiple operations with complications. The incubation period was comparatively long, and there was a delay in starting treatment in at least two of them. In spite of the malignant

4. Woods, A. C.: Am. J. Ophth. **19**:9 (Jan.) 1936.
5. Fuchs, E.: Arch. f. Ophth. **61**:365, 1905.
6. Verhoeff, F. H.: Personal communication to the author.

course in the sympathizing eye, useful vision was attained in the exciting eye in each instance. This was also true in two of the cases in which the condition was unproved.

Such instances tend to substantiate the statements of those who maintain that the inflammation is disproportionately more severe in the sympathizing eye after extraction of cataract and are therefore reluctant to enucleate the exciting eye.[2a] The data for twelve cases in which the condition was clinically diagnosed (table 5) in which neither eye was enucleated are not convincing. While better vision resulted in the exciting eye in five patients, there was a marked difference in only one. In two patients vision in the sympathizing eye was slightly better than that in the exciting eye, and in three patients there was no difference. All in all, the outcome was generally poor in both eyes, only three patients attaining useful vision. While there may be too many extenuating factors to warrant the conclusion that these data indicate the value of enucleation, they do not indicate any disproportion in the severity of the condition in the two eyes, nor do they indicate the value of retention of the exciting eye.

Of seventeen cases of sympathetic ophthalmia following combined extraction of cataract in which the outcome is recorded, there was complete loss of vision in the sympathizing eye in nine (53 per cent) (table 6). Useful vision was attained in eight cases (47 per cent), and in five of these it was 20/40 or better. While this represents the final vision in the sympathizing eye, it does not tell the complete story, for in the three patients in whom the sympathizing eye was enucleated useful vision resulted in the exciting eye. Therefore, eleven patients (64.7 per cent) actually attained useful vision. When thirty-one cases in which the condition was clinically diagnosed are included the data do not show such a favorable result. Considering the whole group of forty-seven cases, useful vision in the sympathizing eye resulted in nineteen (34 per cent). It is probable that both the figures for the cases in which the diagnosis was confirmed and those for the total group lead one to overestimate the damage caused by the inflammation, for in many instances the final vision was undoubtedly influenced by the presence of a cataract which existed before the uveitis appeared.

While no conclusions can be drawn from the data for this group of cases, the results of the study at least indicate that if proper treatment is promptly instituted sympathetic ophthalmia following combined extraction of cataract is not necessarily unusually violent and that the prognosis may not be as hopeless as many authors have indicated.

Sympathetic ophthalmia followed operations for congenital cataract in three patients (table 7). Two were children, and one was 29 years of age. The operations, consisting of a discission in two instances and a linear extraction in one, were all followed by a severe reaction. A

TABLE 3.—*Data for Cases of Sympathetic Ophthalmia Following Combined Extraction of Cataract*

Case	Reference	Age of Patient, Years	Complications	Interval Between Operation and Onset of Sympathetic Ophthalmia	Interval Between Onset of Sympathetic Ophthalmia and Treatment	Interval Between Onset of Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, and Vision, at Onset	Course	Duration	Outcome and Comment
346	Post, L. T.: Personal communication to the author in regard to the cases compiled by D. H. Trowbridge	54	Hyphemia and hypopyon 2 days after operation	10 wk.	1 wk.	1 wk.	Vision 6/12; incipient cataract; plastic iridocyclitis	Moderate	Several months	Vision 6/12; favorably influenced by operation
348	Post, L. T.: Personal communication to the author in regard to the cases compiled by D. H. Trowbridge	?	?	?	?	?	?	?	?	Blind
343	Post, L. T.: Personal communication to the author in regard to the cases compiled by D. H. Trowbridge	?	?	?	?	?	?	?	?	?
353	Morax, V.: Ann. d'ocul. **154**: 705 (Dec.) 1917	73	Followed by iridocyclitis	59 days	1 day	1 day	Iridocyclitis	?	?	Vision 5/10
261	Theobald, G. D.: Am. J. Ophth. **13**: 597 (July) 1930	52	Followed by infection	4 wk.	A few days	6 days	Vision 20/200	Moderate	?	Vision 20/200
262	Theobald, G. D.: Am. J. Ophth. **13**: 597 (July) 1930	56	Extraction of cataract attempted	8 wk.	Within 19 days	19 days	Very severe	Severe	?	?
307	Verhoeff and Irvine [10].....	41	?	10 wk.	12 days	1 wk.	Slight congestion; keratitis punctata; Koeppe nodules	?	1 yr.	Vision 20/30; 1½ yr. after subsidence; extraction of cataract in sympathizing eye 1½ yr. after onset
309	Verhoeff and Irvine [10].....	53	?	8 wk.	8 days	1 wk.	Slight congestion; a few synechiae pupillary exudate; vision 20/50	?	6 mo.	Vision 20/100 8 yr. after subsidence (poor vision due to senile cataract)

310	Verhoeff and Irvine [10]	70	?	8 wk.	30 days	8 wk.	Vision 1/200; slight congestion; keratitis punctata cataract	?	6 mo.	Vision 20/30 1 yr. after subsidence; extraction of cataract in sympathizing eye 6 mo. after onset
305	Woods, A. C.: Personal communication to the author	70	?	2 mo.	3 wk.	At onset	Vision 20/100; involvement of posterior part of uvea	?	?	Vision 20/30
57	Joy [3]	67	Complicated cataract; secondary glaucoma	30 days	Within 3 wk.	19 days	Loss in visual field; iridocyclitis	Moderate	3 mo.	No perception of light
8	Joy [3]	65	?	From 6 to 10 wk.	?	?	?	Severe	?	No perception of light
101	Joy [3]	60	High myopia	3 mo.	?	?	Plastic uveitis with exudate; secondary glaucoma	Severe	?	No perception of light
67	Joy [3]	68	Uveitis	6 mo.	Prompt	1 mo. before	Plastic uveitis	Moderate	18 mo.	No perception of light
238	Wood, D. J.: Brit. J. Ophth. **11**: 217 (May) 1927	53	Abrasion of cornea several days after operation; discission 7 wk. after extraction of cataract	74 days	A few days	Enucleation of sympathizing eye	Iridocyclitis; keratitis punctata	Severe	?	Sympathizing eye enucleated; vision of exciting eye 6/6, 1½ yr. later
232	Schreiber, L.: Arch. f. Ophth. **129**: 127 (Sept.) 1932	66	Complicated cataract; discission 3½ mo. later; secondary glaucoma	6 mo.	Within 2 mo.?	Enucleation of sympathizing eye	Iridocyclitis; secondary glaucoma	Severe	?	Sympathizing eye enucleated; vision of exciting eye 0.5, 2 yr. later
207	Jaeger, E.: Klin. Monatsbl. f Augenh. **78**: 613, 1927	42	Iris in scar; discission 2 mo. after extraction	3½ mo.	2½ mo.	Enucleation of sympathizing eye	Faulty projection; iritis; keratitis punctata	Severe	1 yr.?	Sympathizing eye enucleated; vision of exciting eye 5/8, 1 yr. later
318	Verhoeff and Irvine [10]	68	?	16 wk.	42 days	No enucleation	Immature cataract; keratitis punctata; total synechia	?	6 mo.	Vision of sympathizing eye 20/200, vision of exciting eye 20/50, 1½ yr. after subsidence

subsequent linear extraction was necessary in two patients, and in one of these it resulted in prolapse of the iris and loss of vitreous. The incubation period ranged from seven weeks to six months. The exciting eye was removed and treatment begun promptly in all three cases; in

TABLE 4.—*Data for Cases in Which the Diagnosis Was Confirmed in Which the Exciting Eye Was Not Enucleated*

Case	Complications	Interval Between Operation and Onset	Interval Between Onset and Treatment	Course	Interval Between Onset and Enucleation of Sympathizing Eye	Outcome in Exciting Eye
318	?	16 wk.	42 days	?	Not enucleated	Vision of exciting eye 20/50, vision of sympathizing eye 20/200, 1½ yr. after subsidence
238	Yes	79 days	A few days	Severe	6½ mo.	Vision 6/6, 11¼ mo. after enucleation
232	Yes	6 mo.	Within 3 mo.	Severe	3 mo.	Vision 0.5, 2 yr. after enucleation
207	Yes	3½ mo.	2½ mo.	Severe	1¼ yr.	Vision 5/8

TABLE 5.—*Data for Cases of Clinically Diagnosed Sympathetic Ophthalmia Following Extraction of Cataract in Which Neither Eye Was Enucleated*

Case	Exciting Eye	Sympathizing Eye
132	Perception of light	No perception of light
131	Vision 20/200	No perception of light
111½	Perception of light	No perception of light
105	Perception of fingers	No perception of light
259	Vision 1/60	Perception of light
300	Perception of movements of hands at 4 in.	Perception of movements of hands at 14 in.
338	Perception of light	Vision 4/200
389	Perception of light	Perception of light
42	Vision 6/12	Vision 6/12
33	No perception of light	No perception of light
272	?	No perception of light
335	?	Vision 20/20

TABLE 6.—*Final Vision in Cases of Sympathetic Ophthalmia Following Combined Extractions*

	Number of Cases	Percentage	
20/40 - 20/15	5	29.4	47
20/200 - 20/50	3	17.6	
Perception of light - perception of movements of hands	1	6.0	53
No perception of light	8	47.0	

two of them treatment was started within one day after the onset of sympathetic inflammation. The duration was short, varying from one month to four months. Two of the patients obtained useful vision, and one became blind. In a case of clinically diagnosed sympathetic ophthalmia in which the exciting eye was not removed the outcome

TABLE 7.—*Data for Cases of Sympathetic Ophthalmia Following Operations for Congenital Cataract*

Case	Reference	Age of Patient, Years	Type of Operation	Complications	Interval Between Operation and Onset of Sympathetic Ophthalmia	Interval Between Onset of Sympathetic Ophthalmia and Treatment	Interval Between Onset of Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, and Vision, When Patient Was Seen	Course	Duration	Outcome and Comment
243	Case compiled by the author in the Wills Eye Hospital	29	Linear extraction	Large amount of lenticular cortex; linear extraction repeated 3 wk. later	7 wk.	1 day	1 day	"Mutton-fat" deposits of keratitis punctata; iris congested; vision 6/25	Mild	1 mo.	Vision 6/25; gradual improvement after enucleation
116	Joy [3]	3½	Discission	Hyphemia; exudate in anterior chamber; tension +; no perception of light	6 mo.	Less than 3 mo.	Less than 3 mo.	?	Moderate	2 mo.	Vision 20/40
344	Post, L. T.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	8	Discission	Lens in anterior chamber; glaucoma; lens extracted 1 wk. later, with loss of vitreous and prolapse of the iris	9 wk.	Same day	Same day	Keratitis punctata; pupil contracted	Severe	4 mo.	Blind; enucleation did not influence progress

TABLE 8.—*Data for Cases of Sympathetic Ophthalmia Following Extraction of Traumatic Cataract*

Case	Reference	Age of Patient, Years	Type of Injury	Interval Between Injury and Operation	Type of Operation	Complications	Interval Between Operation and Onset of Sympathetic Ophthalmia	Interval Between Onset of Sympathetic Ophthalmia and Treatment	Interval Between Onset of Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, and Vision, When Patient Was First Seen	Course	Duration	Outcome and Comment
240	van Schievensteen, A., Jr.: Ann. d'ocul. **155**: 392 (Aug.) 1918	31	Perforating wound; foreign body in iris	3 mo.	Extraction of cataract	Patient squeezed at operation, causing prolapse of iris and hyphemia	49 days	3 days	3 days	Vision 0.4; ciliary congestion; tension —	?	6 mo.	Cured
244	Oguchi: Acta soc. ophth. jap. **38**: 98 (June) 1934	39	Blow from piece of wood	?	Extraction of cataract	?	2 mo.	?	?	Iritis and neuroretinitis	?	?	Did not improve
13	Joy [3]	27	Perforation; foreign body in lens	6 yr.	Combined extraction in capsule	Secondary glaucoma and low grade uveitis before operation	7 wk.	A few days	A few days	Vision 20/200; severe iridocyclitis	Severe	5 wk.	Vision 20/15
9 M.	Morax, V.: Ann. d'ocul. **154**: 705 (Dec.) 1917	26	Injury to iris	2 wk.	Extraction of cataract	Prolapse of iris before operation	58 days	Prompt	Prompt	Severe iridocyclitis	Severe	?	Vision 1/25

was poor, with vision of perception of fingers at 2 feet (60 cm.) in the sympathizing eye and loss of perception of light in the eye on which the operation had been performed.

Perforation of the globe at the time of injury had occurred in three of the four cases of sympathetic inflammation following operations for traumatic cataract (table 8). This had resulted in prolapse of the iris in one instance, while in two an intra-ocular foreign body was present at the time of the extraction. The patients were all men from 26 to 39 years of age. The interval between the injury and the operation ranged from two weeks to six years, and with one exception the eye was quiet at the time of operation. In the latter patient low grade uveitis and secondary glaucoma had developed as the result of a perforating wound six years before. Another patient misbehaved during the operation, causing prolapse of iris and a large hyphemia. The incubation period ranged from seven weeks to two months. In at least three of the cases treatment was instituted promptly and the exciting eye enucleated within a few days after the onset. Two of the patients made a satisfactory recovery, and two did badly, while in five cases in which sympathetic ophthalmia was clinically diagnosed three patients attained useful vision.

Of the fifteen cases of sympathetic ophthalmia following operations on the iris, in all but one the condition was attributed to some form of operation for glaucoma (table 9). The exception was a case in which a repair operation was performed for iridodialysis five weeks after a severe contusion. Among the remaining cases, in six the condition occurred after iridectomy, in five it followed operation for iris inclusion, and in three it was due to trephining, while in one case the type of operation was not recorded (table 10). With four exceptions the patients were 40 years of age or older. The incubation period ranged from thirty-three days to four months. Treatment was instituted within two weeks after the onset in twelve cases and within three months after the onset in all. The duration extended from two months to three years. Jaeger [2c] stated that sympathetic ophthalmia does not often follow iridectomy and when it does it is often of a mild type. This was not true in the six cases in this series, for although the exciting eye was removed and treatment begun within two weeks after the onset of the first symptoms, the course was either moderate or severe. Although four patients ultimately attained useful vision, the uveitis was still active in one when the case was reported. In two cases in which the condition was clinically diagnosed the outcome was satisfactory. Peters,[7] in reviewing the reported cases in which sympathetic ophthal-

7. Peters, A.: Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten, Ztschr. f. Augenh., supp. 20, 1936.

TABLE 9.—*Data for Cases of Sympathetic Ophthalmia Following Operation on the Iris*

Case	Reference	Age of Patient, Years	Type of Operation	Complications	Interval Between Operation and Onset of Sympathetic Ophthalmia	Interval Between Sympathetic Ophthalmia and Treatment	Interval Between Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, and Vision, at Onset	Course	Duration	Outcome and Comment
88	Joy [3]	25	Iridodialysis as a repair operation	Operation done 5 wk. after severe contusion	7 wk.	Prompt	Prompt	?	Severe	6 mo.	Vision 20/15—
					Operations for Glaucoma						
341	Higbee, E. H.: Am. J. Ophth. **9**: 584, 1926	42	Iridectomy for secondary glaucoma	Iritis before operation	No sympathetic ophthalmia			No sympathetic ophthalmia	.....	.....	Vision 20/20; no sympathetic ophthalmia at any time
220	Groenouw, A.: Klin. Monatsbl. f. Augenh. **76**: 694, 1926	?	Iridectomy	Prolapse of iris	5 wk.	Within 2 wk.	2 wk.	Disseminated choroiditis; failing vision	Moderate	4½ mo.	Vision, part of 5/15
247	Gifford, S. R., and Lucie, L. H.: Tr. Sect. Ophth., A. M. A., 1926, p. 20	70	Iridectomy	Melanosarcoma of choroid	5 wk.	5 days	5 days	Perception of fingers at 5 in.; rapidly progressive uveitis; exudate in pupil	Severe	?	Vision 6/30
327	Verhoeff and Irvine [10]	61	Iridectomy	?	7 wk.	5 days	1 wk.	Vision 20/50; moderate congestion; many deposits of keratitis punctata; posterior synechia	?	6 mo.	Vision, perception of light; inflammation inactive for 6 mo.
342	Post, L. T.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	60	Iridectomy	Iritis on sixteenth day; hypopyon on twenty-fourth day	33 days	8 days	8 days	Vision 20/30—perception of fingers at 10 ft. 3 days later; tension +; deposits on anterior capsule	Moderate	1 yr. +	Vision 20/20; inflammation still active; progress favorably influenced by enucleation
345	Post, L. T.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	67	Iridectomy	None; low grade iridocyclitis 1 mo. later	7 wk.	2 wk.	2 wk.	Almost total posterior synechia; secondary glaucoma; blurred vision	Severe	8 mo.	Blind; progress not influenced by enucleation; patient died

304	Woods, A. C.: Personal communication to the author	69	Iridectomy	Gaping wound; ectasia of iris; vitreous presenting	11 wk.	1 wk.	1 wk.	Vision 20/15, 1 yr. after onset; involvement of the anterior and posterior part of the uvea	?	3 yr. ?	Subsided
231	Schoenberger, H.: Arch. f. Ophth. **125**: 28, 1930	50	Trephination	Sclerotomy necessary 8 days later; early iridocyclitis	1 mo.	Prompt	Prompt	?	?	?	Total loss of vision
263	Theobald, G. D.: Am. J. Ophth. **13**: 597 (July) 1930	62	Trephination	Iridectomy	15 wk.	4 days	4 days	?	Moderate	?	Vision 20/20
9	Joy [3].....	23	Trephination	Trephination repeated	4 mo.	Less than 3 mo.	Less than 3 mo.	Vision 20/40	Severe	From 3 to 4 yr.	Vision 20/70
249	Gifford, S. R., and Lucie, L. H.: Arch. Ophth. **1**: 468 (April) 1929	A	Iridencleisis	Large subconjunctival prolapse of iris and of ciliary body; part of which was excised later	?	A few days	A few days	Vision blurred; cells; disk blurred	Moderate	?	Vision 20/20—; eye cleared rapidly and remained quiet
347	Post, L. T.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	28	Iridencleisis on right and left eye for secondary glaucoma	None	9 wk.	At onset	6 wk.	Keratitis punctata; cells in anterior chamber; fundus normal; blurred vision	Moderate	2 mo.	Vision 6/4—2; progress favorably influenced by enucleation
314	Verhoeff and Irvine [10]	66	Iridotasis	?	5 wk.	2 days	1 wk. before onset	Vision 20/50; marked congestion; heavy deposits of keratitis punctata; synechiae; vision decreased to 3/200	?	1 yr.	Vision 20/70—(poor vision due to senile cataract); inflammation; inactive 1 yr.
324	Verhoeff and Irvine [10]	44	Iridotasis	?	8 wk.	14 days	Enucleation at onset	Vision 20/40; moderate congestion; a few deposits of keratitis punctata	?	3 yr.	Vision, perception of light; secondary glaucoma; inflammation inactive 1 yr.
330	Verhoeff and Irvine [10]	40	Iridotasis	?	5 wk.	63 days	No enucleation	Vision 1/200; marked congestion; pupillary membrane	.....	3 yr.	Vision in sympathizing eye, perception of light; no perception of light in exciting eye (primary glaucoma); inflammation apparently still active
139	Joy [3].....	62	Operation for secondary glaucoma	?	?	Late ?	?	?	Severe	9 mo.	No perception of light

mia followed trephining operations, mentioned only nine and questioned the diagnosis in several of these. In all three of the cases in this series the condition followed a second operation. Useful vision resulted in two, as well as in two of the three cases in which the condition was clinically diagnosed. Operations for iris inclusion would seem to offer fertile ground for the development of sympathetic ophthalmia, but there are surprisingly few cases reported in the literature in which this condition followed such an operation, and in none of the one hundred and fifty-one cases of sympathetic ophthalmia in the series in New York State which I [3] reported was it the exciting cause. Of the five cases reported here, in three the end-result of the operation was

TABLE 10.—*Data for Cases of Sympathetic Ophthalmia Following Operations for Glaucoma*

Type of Operation	Number of Cases	Outcome	
		Good	Bad
Iridectomy	6	4	2
Trephination	3	2	1
Operation for iris inclusion	5	3	2
Operation for glaucoma	1	0	1

TABLE 11.—*Final Vision in Cases of Sympathetic Ophthalmia Following Operations for Glaucoma*

Vision	Number of Cases	Percentage	
20/ 40 - 2/15	5	33.3	60
20/200 - 20/50	4	26.7	
Perception of light	3	20.0	40
No perception of light	3	20.0	

successful, while in two vision was reduced to perception of light. In a case in which the condition was clinically diagnosed which followed bilateral iridotasis, final vision in one eye was 20/20 and in the other was reduced to perception of shadows.

Obviously no conclusions can be drawn regarding the relationship of the type of operation and the ultimate outcome. However, in this series sympathetic inflammation due to iridectomy for glaucoma was not mild or apparently infrequent.

The outcome was generally more favorable when treatment was started promptly. Among twelve patients for whom treatment was instituted within two weeks after the onset of sympathetic inflammation, useful vision resulted in eight, and four became blind, while in three patients in whose cases there was a delay of six weeks or more, one attained useful vision, and two became blind. Of the fifteen proved

cases in which the condition followed operations for glaucoma (table 11) the outcome in nine (60 per cent) was satisfactory, while in six (40 per cent) it was poor. The final vision in several instances was unfavorably influenced by the presence of primary glaucoma or senile cataract.

SECONDARY OPERATIONS

Because of the great frequency of cataract and glaucoma in both eyes, irrespective of the uveitis, subsequent operations to improve vision would seem to be more often necessary in sympathetic inflammation due to operation than in that due to other causes. For the same reason the prognosis would appear to be less favorable. While the exciting eye tolerates operations well, the sympathizing eye is notoriously sensitive, even after a long period of quiescence. Woods [8] found that pigment therapy immediately preceding the operation is of real value in diminishing the reaction and in promoting the healing. Verhoeff [9] has shown that if the patient is kept under the influence of diphtheria antitoxin the eye can sometimes be safely operated on even before the subsidence of inflammation. However, he and Irvine [10] warned that if the patient cannot be made sensitive to the antitoxin and if the treatment is not obviously beneficial, surgical intervention should be avoided.

Table 12 shows the data for those cases of postoperative sympathetic ophthalmia in which either the exciting eye or the sympathizing eye was operated on during or subsequent to the inflammation. In nine of the patients the exciting cause was extraction of cataract, and in two the condition was attributed to iridectomy. The sympathizing eye had already been removed in all four patients whose exciting eyes were operated on. The surgical intervention, consisting of discission in three instances and of iridotomy in one, was well tolerated in all the cases and resulted in improved vision. The surgical intervention on the sympathizing eye was naturally more complicated, and in three patients more than one operation was performed, despite which there was an actually unfavorable outcome in only one case. The three cases in which the diagnosis was confirmed in which the sympathizing eye was operated on were Verhoeff's, whose unusually good results with diphtheria antitoxin were reported in 1935.[10]

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8. Woods, A. C., quoted by de Schweinitz, G. E.: Tr. Ophth. Soc. U. Kingdom **46**:257, 1926.

9. Verhoeff, F. H.: Arch. Ophth. **56**:28 (Jan.) 1927.

10. Verhoeff, F. H., and Irvine, S. R.: New York State J. Med. **36**:63 (Jan. 15) 1936.

TABLE 12.—*Data for Cases of Postoperative Sympathetic Ophthalmia in Which Either the Exciting Eye or the Sympathizing Eye Was Operated On*

Case	Reference	Age of Patient, Years	Exciting Cause of Sympathetic Ophthalmia	Type of Secondary Operation		Time of Second Operation	Effect of Operation	Outcome
				Exciting Eye	Sympathizing Eye			
			Cases in Which the Diagnosis Was Confirmed					
232	Schreiber, L.: Arch. f. Ophth. **120**: 127 (Sept.) 1932	66	Extraction of complicated cataract	Discission	.....	1 yr. after subsidence	Favorable	Vision 0.5
307	Verhoeff and Irvine [10].....	41	Extraction of cataract	.....	Extraction of cataract	6 mo. after subsidence	Favorable	Vision 20/30
310	Verhoeff and Irvine [10].....	70	Extraction of cataract	.....	Extraction of cataract	At subsidence	Favorable	Vision 20/30
327	Verhoeff and Irvine [10].....	61	Iridectomy	.....	Operation	?	Unsuccessful	Vision, perception of light
			Cases in Which the Condition Was Clinically Diagnosed					
258	.....	..	Extraction of cataract	Discission	.....	?	Favorable	Useful vision
275	Hambresin [2a]	?	Extraction of cataract	Discission	.....	Shortly after subsidence	Favorable	Useful vision
105	Joy [3]	Adult	Extraction of cataract	Iridotomy	.....	Shortly after subsidence	Favorable	Patient able to get about
252	Vejdovsky, V.: Oftal. Sborn. **7**: 109, 1932	?	Extraction of cataract	.....	Preliminary iridectomy; extraction of cataract; capsulotomy	?	Comparatively favorable	Vision, perception of fingers at 40 cm.
274	True, H., and Dejean, C.: Bull. Soc. franç. d'opht. **38**: 646, 1925	72	Extraction of cataract	.....	Preliminary iridectomy; extraction of cataract	About 2 mo. after subsidence	Comparatively favorable	Vision, counting fingers
260	Cases compiled by the author in the Wills Eye Hospital	8	Linear extraction combined with iridectomy	.....	Capsulotomy; paracentesis; iridectomy	During course of uveitis	Comparatively favorable	Vision, perception of fingers at 1.5 meters
333	Verhoeff and Irvine [10].....	45	Iridectomy	.....	Iridectomy	?	Favorable	Vision 20/20

SUMMARY

Sympathetic ophthalmia is too complicated a disease to permit one to draw conclusions from such a small number of cases. However, study of this series brings out several points worthy of notice.

1. The final visual results indicate that the prognosis of postoperative sympathetic ophthalmia is not necessarily as unfavorable as many authors have stated, provided proper treatment is instituted with promptness.

2. The final outcome in the cases in which the condition followed combined extraction of senile cataract was less favorable than in those in which it followed other intra-ocular operations.

3. The inflammation in the sympathizing eye was disproportionately more severe than that in the exciting eye in three cases of sympathetic ophthalmia due to extraction of cataract in which the diagnosis was confirmed and three cases of the same kind in which the condition was clinically diagnosed. However, this was not apparent in twelve cases in which the condition was clinically diagnosed in which neither eye was enucleated.

4. There is no indication that sympathetic ophthalmia due to iridectomy for glaucoma is particularly rare or that its course is mild.

5. The results in the few instances in which secondary operations were performed indicate that the exciting eye tolerates surgical intervention well and that the sympathizing eye can often be safely operated on if it is properly prepared for the intervention.

504 State Tower Building.

Alan C. Woods presented the facilities of the library of the Wilmer Ophthalmological Institute for use in making this study.

OPTIC ENCEPHALOMYELITIS

REPORT OF A CASE

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Optic encephalomyelitis is comparatively rare. The object of this paper is to report another case of this disease that presented features of special interest.

REPORT OF CASE

History.—Mrs. R. O., aged 34 years, came to me on Nov. 20, 1932, complaining of a feeling of pressure and of loss of vision in the right eye, of about two weeks' duration.

There was no history of diseases of childhood, but the patient had pneumonia five years previously and had had occasional sore throat. She suffered from dyspnea on exertion; swelling of the ankles, especially when on her feet for any length of time, and occasional dizzy spells. Her gallbladder had been removed nine years before, but she still complained of gas and of discomfort in the abdomen and epigastrium after meals. There was no history of jaundice. The family history was not important.

Physical Examination.—The patient was sent to the Royal Victoria Hospital, of Montreal, on November 22, for a complete physical examination. This showed chronically infected tonsils and recession of the lower mandible. The liver and spleen were within the normal limits, and the cardiovascular system was normal. Examination of the respiratory system revealed showers of fine moist crackles, which did not clear when the patient coughed. The abdomen was soft, but there were no areas of tenderness and no abnormal masses. The blood count showed no abnormalities; the calcium and phosphorus contents of the blood were normal. The Wassermann tests of the blood and the cerebrospinal fluid were negative. The urine showed a few hyaline casts, and epithelial cells; albumin and a few white corpuscles were present. Roentgen examination gave negative results, as did examination of the pelvis and of the nervous system.

Examination of the Eyes.—Right Eye: The lids were normal and free from signs of irritation. A scar of the cornea extended from the nasal side to and over the greater part of the pupillary area, the result of phlyctenular disease in childhood. The pupil (4 mm. in diameter) reacted to light and in accommodation; the media were clear. Vision was counting fingers uncertainly at 1 foot (30.5 cm.). The field of vision could not be taken on account of the reduced sight, but on rough tests there seemed to be some defect in the nasal field. The ocular movements were normal; the tension was normal, and the fundus showed no changes.

Left Eye: The cornea was clear; the pupil was circular and active. Vision was 6/6. The field of vision was full; the fundus was normal, and ocular movements were normal.

Subsequent Examinations.—The patient was discharged from the hospital on November 28 but was readmitted on December 11, at which time the sole complaint was failing sight in the left eye, which had developed within the course of an hour or so.

Examination of the left eye showed the pupil to be dilated and inactive. Vision was perception of movements of the hand only. There was an apparent defect in the central field, as shown with tests with very large objects. The tension and the fundus were normal. Examination of the right eye showed the pupil to be dilated and inactive. Vision was counting fingers at 6 inches (15.2 cm.); the fundus was normal.

The following examinations gave negative findings: neurologic examination, dental examination, pelvic examination, tests of the cerebrospinal fluid, the Wassermann test of the blood, and roentgen examination of the skull. The heart and lungs were as previously reported. The blood count revealed 4,020,000 red cells, 3,650 white cells and 80 per cent hemoglobin. There were leukopenia, neutropenia, more immature forms of polymorphonuclears than mature forms and relative monocytosis. The agranulocytic character of the blood picture, the high lymphocyte count (relative) and monocyte count and the shift of the hemogram to the left demonstrated a marked myelotoxic process. The total number of the white cells was diminished.

Course.—During the patient's stay in the hospital there was a variation in her vision. The sight of the right eye ranged from counting fingers at 6 feet to 20/200. Then it declined to counting fingers at 4 feet (121.9 cm.). The vision of the left eye deteriorated from counting fingers to perception of movements of the hand. Then it decreased until there was no perception of light and remained so until the patient's death. The patient was discharged from the hospital on Jan. 28, 1933. The diagnosis was retrobulbar neuritis and secondary atrophy of the optic nerve.

On April 5 she was admitted to the neurologic service of the Royal Victoria Hospital under Dr. Russell on account of failing vision, a feeling of epigastric tightness and burning pain and pressure in the chest. She gave the following history:

About two weeks after leaving the hospital a burning pain suddenly occurred one night in the entire right leg; the feeling was as though the entire leg had something very hot applied to it. This feeling lasted a few days and then passed. A few days later a peculiar feeling of extreme tightness accompanied by a burning pain occurred across the upper part of the abdomen. While this burning pain was present the patient could not endure to have the bedclothes or other materials touch the skin of the affected regions. On a few occasions she noticed that when she touched the leg a peculiar sensation passed through it, as though an electric shock was being felt. A week or so after the pain first appeared she noticed that when she scratched the leg there was no sensation. The area of loss of sensation had progressed upward until it reached about the level of the fourth rib, anteriorly. Just before her admission to the hospital she became very constipated; she attributed this to the fact that she could not produce any pressure in the abdomen with the muscles.

Neurologic Examination.—A neurologic examination by Dr. Young revealed evidence of a lesion of the optic nerves and of the spinal cord. The other cranial nerves were normal. The mental status and speech were normal. There was no abnormality, subjective or objective, of the upper extremities, whereas the lower extremities, especially the right leg, were very weak. Paralysis of the right leg

was almost complete; the patient was capable of only slight movement of the foot and toes. Movement of the left leg was practically full but was weak. The tone was slightly increased in the right leg. No atrophy and no fibrillation were present. Coordination could not be tested accurately.

A lesion at the level of the fifth dorsal segment of the spinal cord on the right side was revealed by sensory examination. Touch was identified each time with possibly some impairment of the fifth lumbar segment on the right. There was loss of the sense of pain on the left side up to the seventh dorsal segment, just at the lower crease of the breast, where pain was felt as extreme. The sense of pain was impaired on the right over an area corresponding to the third to the tenth dorsal segment behind, and the fourth to the seventh dorsal segment in front. The areas of the disturbance of the sense of heat and cold corresponded to those of disturbance of the sense of pain. The sense of vibration was impaired in the right leg, and vibration was identified in the left leg. It was felt better at the sixth or seventh dorsal segment below. The sense of position was impaired in the right toe. The test with figure writing was not reliable. The cerebrospinal fluid showed an increased amount of protein (Pandy test, plus) and 40 lymphocytes per cubic centimeter. The Wassermann reaction was negative. This suggested Brown-Séquard's paralysis of the fifth dorsal segment on the right, and a diagnosis of encephalomyelitis was offered.

Final Examination of the Eyes and Subsequent Course.—The optic disks were pale and atrophic. Total blindness was present in the left eye. The patient counted fingers with the right eye at 8 inches (20 cm.).

On June 9 the patient asked to be discharged from the Royal Victoria Hospital with a view to going to New York for consultation, where she was admitted to the Mount Sinai Hospital on July 2. While there she contracted bronchopneumonia, and died on July 10.

Report of Postmortem Examination.—A postmortem examination confirmed the clinical findings of encephalomyelitis. The report of the postmortem examination from the Mount Sinai Hospital is as follows:

Coronal sections of the brain through the optic chiasm and tracts revealed on microscopic examination deposits of fat in the optic tracts and some demyelinization through the chiasm. Many of the smaller vessels in this and other regions showed dilated perivascular spaces and cellular infiltrations. The spinal cord revealed patchy areas of demyelinization and deposits of fat in various tracts at different levels. There was mild cellular infiltration of the meninges. On gross examination the dura was normal. The subarachnoid space was increased and was distended with clear fluid. The brain was softer than normal and injected. The convolutions were prominent, particularly in the region of the sylvian fissure. The sulci were correspondingly deepened. The arteries showed a mild degree of atherosclerosis. The ears and sinuses were normal. The eyes were removed (no report was made). The spinal cord was softer than normal and somewhat compressed along its entire length anteroposteriorly. Section of the brain revealed no alterations visible to the naked eye. Section of the spinal cord showed marked softening and disorganization of the mid-dorsal segments, with reduction in size of the upper dorsal segments. No other changes were seen with the naked eye.

The cause of death was acute encephalomyelitis of the brain and spinal cord, subacute meningo-encephalomyelitis and bronchopneumonia of the lower lobes of both lungs. There were fibrous pleural (in the right lung) and pleuropericardial adhesions; hydrothorax of the left lung; total adherent pericardium; necrotizing

cystitis; hydro-ureters and hydronephrosis (slight); ascending pyelonephritis; parenchymatous degeneration of the heart and liver; acute infectious splenic swelling, and scars of an old operation—cholecystectomy.

SUMMARY

Optic encephalomyelitis occurred in an otherwise healthy woman 34 years of age. There were rapid loss of vision of the left eye, with no recovery, and loss of vision of the right eye, with partial recovery. Paralysis of the right leg and of the abdominal muscles followed about four months after the onset of the disease in the optic pathway. The feature of special interest in this case is the long interval (four months) during which the course of the ocular phenomena remained obscure until the definite signs presented themselves in the spinal cord and thus completed the picture of optic encephalomyelitis. A further interesting finding was marked leukopenia, which was present during the entire course of the illness.

The Mount Sinai Hospital authorities furnished the report on the autopsy.

SCLEROMALACIA PERFORANS

REPORT OF A CASE

SAMUEL P. OAST, M.D.
NEW YORK

In the January 1934 issue of the ARCHIVES Dr. J. van der Hoeve,[1] of the university of Leyden, published a report of a series of four cases under the title of scleromalacia perforans. During the course of this report he stated that so far he had been unable to find any published account describing a similar condition and therefore concluded that his cases were the first recorded instances of a condition which he described elsewhere as follows:

A small slightly elevated inflammatory nodule appeared under the conjunctiva at a distance of about 10 mm. from the limbus. This nodule underwent necrosis at the center, discharged a yellowish cheesy material and then disappeared, leaving the deep blue color of the uvea in evidence beneath. This process repeated itself and showed a tendency to travel around the circumference of the eye in ring fashion and exhibited no disposition to involve the cornea or the anterior part of the uvea.

The case I am reporting appears to fit into this picture closely.

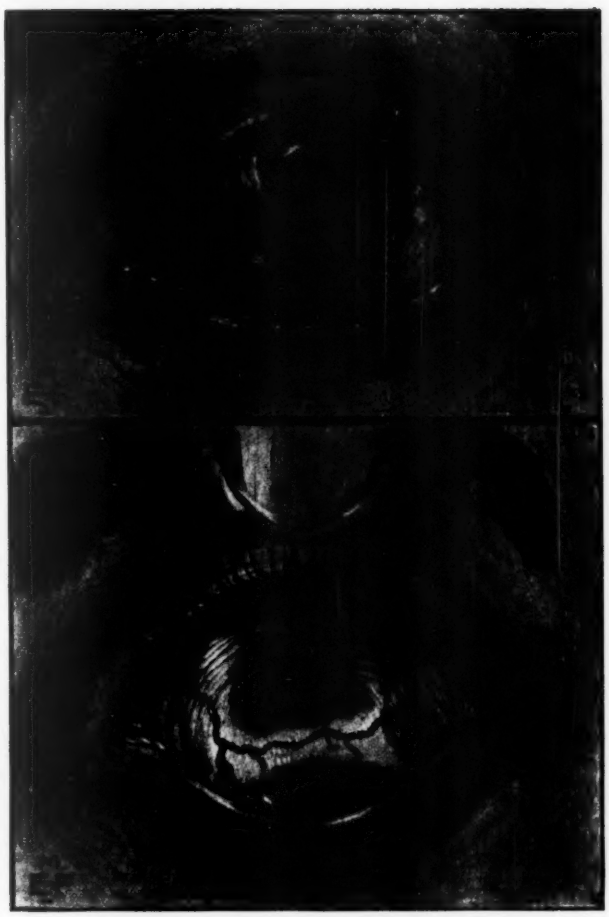
The patient was a man from Dr. Shine's clinic at the New York Eye and Ear Infirmary, 76 years of age, of robust appearance, who gave no history of previous serious illness, and did not recall ever having had any symptoms suggestive of rheumatism or arthritic involvement. I should like to emphasize this fact particularly, because in three of the four cases which van der Hoeve reported polyarthritis was a conspicuous accompaniment of the scleral picture.

The patient was first seen in the clinic about two years ago, at which time he came because of having been struck on the eye now affected (the left eye) by a chip while chopping wood. This blow was evidently severe, not judged so much by the external evidence (for all that could be found was a small laceration of the conjunctiva from 4 to 5 mm. long and just temporal to the limbus) but because of an extensive hemorrhage into the vitreous, reducing vision to perception of movements of the hand and precluding all efforts at examination of the fundus. It was thought that the sclera had not been perforated on this occasion because the tension remained normal and a roentgenogram taken to detect an intra-ocular foreign body showed no abnormality.

A few weeks after this injury the eye became quiet to all appearances. However, it was three months before the vitreous cleared enough to permit satisfactory examination of the fundus, when it was found to present no important changes. By this time visual acuity had returned to 20/30 without a correcting lens.

Read before the Section of Ophthalmology of the New York Academy of Medicine, Jan. 18, 1937.

1. van der Hoeve, J.: Scleromalacia Perforans, Arch. Ophth. **11**:111 (Jan.) 1934.



Scleromalacia perforans.

UNIV. OF MICH.

The eye remained quiet until about eight months ago (fifteen months after the injury which prompted the patient's first visit to the clinic), when there appeared, at about from 6 to 7 mm. from the limbus and just over the point of insertion of the superior rectus muscles, an inflammatory nodule approximately 5 mm. in diameter. This nodule was slightly painful and red, with a yellowish necrotic center, and appeared to be deeply seated under the conjunctiva. It was thought to be an abscess and was accordingly incised, but no pus was found, only caseous material being evacuated. After a few weeks this nodule had disappeared, and it could be seen that the underlying sclera had been virtually destroyed, leaving the bluish uvea in plain view beneath, except for a thin covering of translucent cicatricial tissue. While the original nodule was in the process of resorption a similar nodule appeared a short distance from the first, and this cycle has been repeated consecutively, the lesions traveling counterclockwise, until now fully two thirds of the eye's circumference has been traversed.

There has been a tendency for these foci to coalesce in places, leaving rather large areas where loss of scleral substance may be noted. The cornea has remained clear throughout, and there has been no sign of active involvement of the anterior part of the uvea. However, the opacities in the vitreous have increased so that no clear view of the fundus may be obtained at the present time. Vision is now reduced to the ability to see movements of the hand only, presumably because of the opacities in the vitreous.

I believe this to be a very rare condition, for, aside from the report of van der Hoeve's four cases, I have been unable to find any other mention of the condition in the "Index Medicus" or the "Quarterly Cumulative Index," though possibly it has been reported under another name. Most of my colleagues to whom I have shown the patient have never seen its counterpart before.

As van der Hoeve gave no suggestion as to the etiology of the condition in his cases, my associates and I naturally became interested in the condition from that standpoint. Because of the apparently low grade inflammatory process with termination in the formation of caseous material, it immediately suggested tuberculosis. Therefore on three separate occasions we inoculated an albino rabbit with caseous material obtained from the foci, twice in the anterior chamber and once under the conjunctiva, but without producing any comparable pathologic process or, for that matter, any noteworthy inflammatory reaction.

Previously the usual laboratory examinations, including smears and cultures, were done; these yielded no organisms aside from the normal inhabitants of the conjunctival sac.

The Wassermann reaction was negative, and the blood sugar content was 100 mg. per hundred cubic centimeters. General physical examination revealed nothing of moment. A search for distant foci, including roentgen examination of the teeth, nose and throat, showed nothing unusual, and a dark field examination for Spirochaeta pallida gave negative results. A biopsy was done and showed only granulation tissue and cellular débris. No tuberculin tests were carried out, because

it was thought that the negative results obtained in the repeated inoculation of rabbits had excluded direct tuberculous infection, and tuberculous allergy was not seriously considered because of the appearance and behavior of the lesions. Perhaps these tests should have been carried out also.

At no time was any appreciable involvement of preauricular or any general febrile reaction detected. The appearance of the lesions, however, did suggest a similarity to those found in squirrel plague or in conjunctivitis necroticans, and with this in mind my associates and I, with the help of Mr. Burchell, tried to induce a growth of fungi on glycerin agar (having previously tried the ordinary stock mediums). We also made efforts to produce a growth in the absence of oxygen, but our efforts in these directions, as in others, proved unavailing, and we know no more about the etiology now than when we started.

The term scleromalacia is suggestive of some analogy between this condition and keratomalacia. Naturally, one thinks at once of vitamin deficiency as a producing factor, and the condition doubtless will bear more investigation along these lines. However, if this were the case one would expect the cornea or the fellow eye to become involved, and I should like to emphasize at this point that neither eye has presented any clinical evidence of pathologic changes at any time within the period of our observations.

The obvious predilection of the process for the sclera and the freedom enjoyed by the usually more vulnerable structures, the uvea and cornea, in spite of their proximity, suggest to some (Wood and others [2]) a disturbance of the calcium metabolism, and it would not have been amiss to have a determination of the blood calcium content made during the active stage of the disease, but it did not occur to me at that time.

I cannot help but believe that some connection exists between this condition and the patient's previous injury, but in what manner or to what extent is extremely problematic.

At no time has any rise of the intra-ocular tension been observed, but even with normal tension it is difficult to explain why there has been no tendency to staphyloma when the uvea appears to be almost the sole retaining tunic.

Our treatment has been symptomatic and varied. The one thing which seems to have exerted a beneficial influence was irradiation with ultraviolet rays administered by means of a Birch-Hirschfeld carbon lamp with a uviol filter. After the first few exposures the eye became appreciably quieter, and the pain lessened. There has been continued

2. Wood, D. J.: Calcium Deficiency in the Blood with Reference to Spring Catarrh and Malignant Myopia, Brit. J. Ophth. **11**:224 (May) 1927.

improvement ever since, the eye at present being practically free from inflammatory symptoms. Just how much the ultraviolet therapy had to do with the improvement it is hard to say, but no doubt exists in my mind or in that of the patient that the course of the disease took a definite change for the better with the beginning of these exposures.

Van der Hoeve, in discussing the condition, stated that he had chosen the name scleromalacia in preference to scleritis necroticans, as had been suggested, because of the relative freedom from inflammatory signs, but in the case reported here there has never been any question in the minds of those who saw the condition in its more active stages that a true inflammatory reaction, though relatively low grade, was in progress. At any rate, the picture would not correspond clinically to my understanding of primary degeneration.

It looked, at one time, as though we were going to secure a pathologic specimen, which perhaps would have shed some light on the etiology, but, owing to the efficacy of the ultraviolet rays or, perhaps, to the termination of a natural course, that possibility has become remote.

ORGANIZATION OF THE DEPARTMENT OF OPHTHALMOLOGY OF THE LONG ISLAND COLLEGE OF MEDICINE

JOHN N. EVANS, M.D.
BROOKLYN

The department of ophthalmology of the Long Island College of Medicine [1] has inaugurated what is probably a new method for the training of young men for the specialty of ophthalmology.

In order to appreciate this method, which may be called supplementary fellowship, it is necessary that the general plan of organization of the department be outlined.

The clinic and hospital, though theoretically separate, are for the most part staffed by the same physicians, and in practice are nearly identical. All the members of the staff aid in teaching undergraduates, though only a few hold teaching appointments.

There are a director and an assistant director of the dispensary, who are also the ophthalmologist in chief and the associate ophthalmologist, respectively, to the hospital. They represent the college as professor and clinical professor, direct policies and maintain coordination. The work in the dispensary is arranged as follows: Division A serves Monday, Wednesday and Friday, and division B serves Tuesday, Thursday and Saturday.

Each division has a chief of clinic, who is directly responsible for the management of the cases in his division. Each of the remaining members (sixteen) of the staff has a maximum of six cases assigned to him each day in the clinic (three new cases and three old cases). He studies these cases carefully, any necessary advice or assistance being given by the chief of clinic. The interns, residents and fellows are assigned cases the same as other members of the staff of the dispensary. Should the case be referred to a special consultation clinic (the clinic for patients with glaucoma, the clinic for patients with squint, the clinic for perimetry, the general medical clinic or the neurologic clinic) or should the patient be admitted to the hospital for operation or study, his physician must keep in contact with him and must be present with him on every possible occasion. It is only through such

1. The Long Island College Hospital and the dispensary work in conjunction with the Long Island College of Medicine.

personal study and follow up that the younger physicians can be trained in the proper management of cases in private practice.

Members are first trained in cadaver surgery, then in minor surgery and finally in major surgery. As their aptitude becomes more evident they are eligible for promotion. Seniority of service alone plays a minor part in promotion. Aptitude and genuine interest in ophthalmology are the determining evidence of fitness for advancement.

The internships are open primarily to young physicians who plan to practice in Brooklyn, who have been graduated from a grade A school and who have served an internship in a general hospital—in a medical or rotating service in an institution approved by the American Medical Association. Applicants are accepted after a personal interview by the ophthalmologist in chief. There are no formal examinations.

The internship in ophthalmology (it is termed a residency in ophthalmology by the Council on Medical Education and Hospitals of the American Medical Association) is for one year and follows a definite program of hourly duties and instruction. Assignments are listed in a formal program arranged for each month, and detailed duties, obligations and methods are set forth in the constantly available manual of the hospital.

The residency in ophthalmology is open to the intern after a satisfactory service in this institution (or its equivalent, as determined by the ophthalmologist in chief). The residency is for one year and is likewise open to those who anticipate practicing in Brooklyn. The resident follows a program of duties and instruction similar to that which is followed by the intern.

The fellowships—two in number at present—are open only to young men who have completed the internship and residency just outlined (or its equivalent, in the opinion of the ophthalmologist in chief). They must have opened their private offices in Brooklyn and must maintain active practice in Brooklyn in ophthalmology at least three days a week. The alternate three days are devoted to their fellowship work. The stipend for the fellowship is (at present) $1,200 a year, but this amount may be varied at the discretion of the ophthalmologist in chief.

The primary purpose of the fellowship is to round out the training of these young physicians so as to prepare them adequately for the examinations of the American Board of Ophthalmology. In some instances it may be advisable to limit a fellowship to one year; in other instances it may be necessary to continue the fellowship two, or even three, years. The fellow assists in the instruction of interns, residents and undergraduate medical students and receives instruction and training by means of an assignment of hourly duties as arranged by a scheduled

monthly program. He agrees to accept appointment to the staff of the dispensary on the completion of his fellowship. In this way physicians of the school's own training staff the dispensary and, later, the hospital.

Besides the obviously continuous duties as set forth in the sample program, the fellows, residents and interns receive progressive instruction each month between 10 and 12 a. m. and between 3:30 and 5 p. m. For instance, courses in anatomy and embryology are conducted by the department of anatomy of the college; courses in pathology of the eye are given under the auspices of the department of pathology of the

Program for Fellows and Residents, November 1936 *

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
8- 9	Rounds	Check records	Check records	Rounds	Check records	Check records	Free
9-10	Refraction clinic	Rounds	Rounds	Refraction clinic	Rounds	Rounds	Rounds
10-11	Refraction clinic	Anatomy	Anatomy	Refraction clinic	Anatomy	Anatomy	Free
11-12	Refraction clinic	Anatomy	Anatomy	Refraction clinic	Anatomy	Anatomy	Free
12- 1	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch
1- 2	Clinic	Clinic	Clinic	Clinic	Clinic	Clinic	Free
2- 3	Clinic	Clinic	Clinic	Clinic	Clinic	Clinic	Meet relatives
3- 4	Glaucoma clinic	Operating	Staff rounds	Glaucoma clinic	Operating	Staff rounds	Free
4- 5	Staff rounds; instruction in refraction	Staff rounds; instruction in refraction	Instruction in refraction	Staff rounds; instruction in refraction	Staff rounds; instruction in refraction	Open	Free

* Time for attendance at neighboring seminars, courses and society meetings is arranged.

hospital, and instruction in optics is conducted by the departmental optician. There are special courses under the department of physiology, the department of neurology and the department of medicine, and the young physicians are required to take part in, or initiate, research work. Special courses are given in various branches of ophthalmology, not only during the hours spent at the clinic but also regularly from 3:30 to 5 p. m. throughout the year.

Examinations and quizzes, assignments from texts and the reading of periodical literature are requisites. The fellows must write a preliminary report at the end of the first six months and a thesis at the end of the first year.

Clinical Notes

CONGENITAL NYSTAGMUS

L. Pellman Glover, M.D., Altoona, Pa.

The recent report of Cox [1] on congenital head nodding and nystagmus has led me to question more closely in cases of nystagmus the history of other members of the family in an attempt to trace a hereditary element, even though no head nodding was present. I was extremely fortunate in obtaining the genealogy of four generations through the efforts of the mother of one of my patients with nystagmus. The history may be relied on, as the mother is very intelligent and closely questioned other members of the family.

She volunteered the information that the women never had the trouble, but seemed to transmit it to the males. She also mentioned that by some coincidence the fourth born, if a male, had the trouble

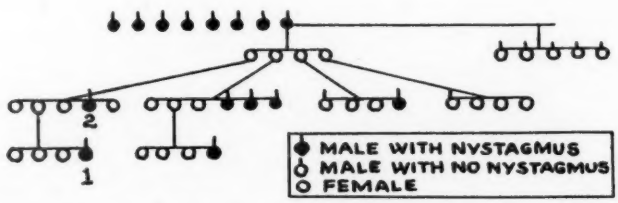


Diagram of genealogy of four generations of the family showing hereditary nystagmus.

which the earlier members escaped. To quote from her own statement, "My grandfather's eyes were crossed and roving. He had seven brothers, all of whom had similar eye trouble. They must have made a pretty picture at the table. Grandfather had five sons and four daughters who were normal. Each daughter, with one exception, had one or more children with roving eyes. Some had nodding of the head or held the head back. The eyes always roved, but the nodding or holding the head back usually stopped when they grew older."

The family tree is shown in the diagram. The mother of the member numbered 1 gave the information. Only the members numbered 1 and 2 have been examined by me. The findings in their cases are reported.

REPORT OF CASES

Case 1.—The patient was C. B., aged 4 years. The family noticed that both eyes showed a rotatory nystagmus since birth. As soon as the child began to

1. Cox, Ronald A.: Congenital Head-Nodding and Nystagmus, Arch. Ophth. **15**:1032 (June) 1936.

walk the parents noticed that he held his head down and looked up in order to see straight ahead. There was no injury sustained at birth.

Examination showed an apparently normal and mentally alert child. He held his head down and looked up to see, as a person would over the top of a pair of glasses. An equal rotatory nystagmus was present, but there was no squint. Aside from this all the findings were normal. On refraction a —1.00 sphere was ordered for the right eye and a —1.25 sphere for the left. Examination five months later, in November 1936, showed an improvement in the nystagmus and in the tilting of the head.

CASE 2.—H. W., aged 17, an uncle of the first patient, was first seen in May 1934. He had been wearing glasses for five years. Nystagmus had been present from birth, and the right eye had always turned in.

Examination showed normal holding of the head. There was a rotatory nystagmus of both eyes, with a convergent squint of 35 degrees of the right eye. The pupils were dilated and reacted poorly to light. Both optic nerves showed a slight gray atrophy. Refraction with the eyes under the influence of homatropine showed vision in the right eye of 6/30 with a + 1.50 sphere + 3.25 cylinder, axis 90, and the patient could read Jaeger's test type 10. Vision in the left eye was 6/21 + with a + 1.50 sphere + 3.50 cylinder, axis 105, and the patient could read Jaeger's test type 10.

The patient was instructed to carry out fusion exercises and in two months was able to fuse satisfactorily with the eyes straight. There was no change in the nystagmus. When he was last seen, in August 1935, the condition had remained stationary. The patient has always been in perfect health and is mentally alert.

COMMENT

While in the cases just reported the patients did not show the typical head nodding, the first patient evidently held the head back to compensate for the nystagmus. I feel that the condition just described would come under Cox's classification of head nodding and nystagmus. Unfortunately, other members of the family could not be studied, as they live too far away. In any event, the family history of hereditary nystagmus is evident. The unusual feature is the apparent transmission through the females, who did not have the disease, to the males, who were susceptible.

A NEW HOLSTER FOR THE OPHTHALMIC WARD AND CLINIC

SAMUEL PELUSE, M.D., CHICAGO

The ophthalmoscope holster here described was designed in the department of ophthalmology of the Cook County Hospital, where it is now in daily use by the interns and resident physicians.

Its practical features have been fully attested to by those who have used it in the routine examination of patients in the wards and clinics.

The holster eliminates the loss of time and patience attendant on the misplacing of the ophthalmoscope and other accessories, such as a pencil or pen, a rule, a retinoscope or a pocket flash-light during rounds of the wards. It dispenses with the disarrayed and frayed pockets encountered when the ophthalmoscope is carried in the usual manner on one's person. The continual hazard of "hooking the chair" or other objects when carrying the ophthalmoscope in the rear pocket

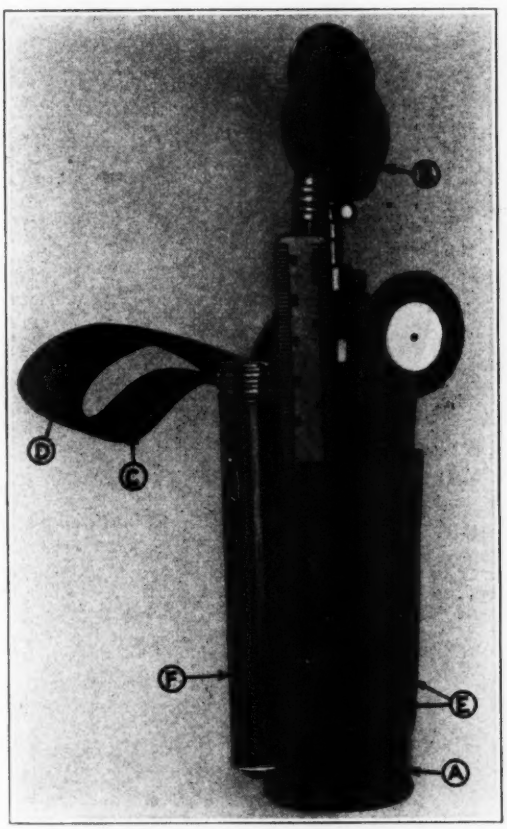


Fig. 1.—The Peluse ophthalmoscope holster in detail.

of the trousers is eliminated. The danger of damage to the ophthalmoscope by accidentally dropping it on the floor is done away with.

During the short period that it has been used (the past six months) the cost of maintenance of the ophthalmoscopes, as verified by the purchasing department, has dropped to a minimum.

The device (fig. 1) is made of leather and weighs 3 ounces (85 Gm.). It consists of a sleeve (*A*) 5 inches (12.7 cm.) in length and 1¾ inches (4.5 cm.) in diameter, open at the top and closed at the bottom for ensheathing the battery case of the ophthalmoscope (*B*). This sleeve is supported by a strap (*C*) made of stiff leather 1½ inches

(3.8 cm.) wide and 11 inches (28 cm.) long which is stitched to the back and fitted with a snap catch (*D*) for straddling the wearer's belt.

On each side, on the outside of the sleeve is a pocket (*E*) 1 inch (2.5 cm.) wide and 3 and ¾ inches (9.5 cm.) long, used as a receptacle for the retinoscope, pencil and flexible rule. The pocket flash-light (*F*), as well as the pen (if it is used), may be supported by slipping the clip over the free edge of the sleeve or over the edge of one of the side pockets.

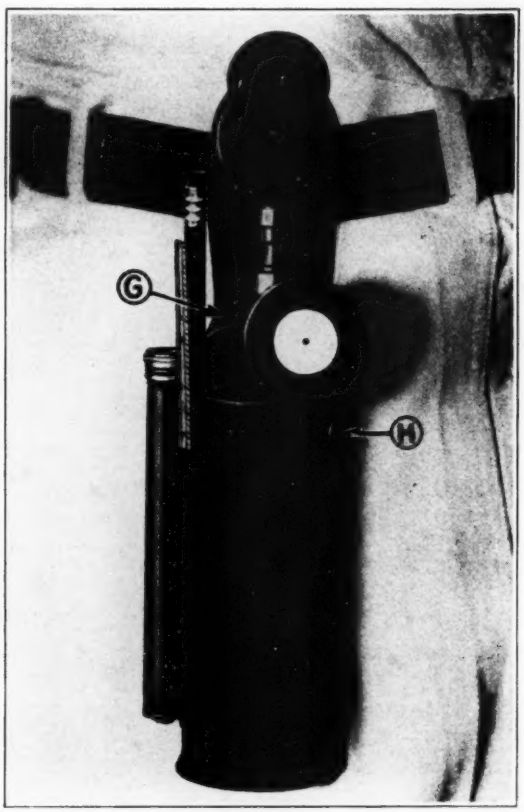


Fig. 2.—The Peluse ophthalmoscope holster strapped to the wearer's belt.

A thin strap of leather (*G*) ½ inch (1.2 cm.) wide and 5 inches (12.7 cm.) long (fig. 2) is sewed to the supporting strap and allowed to curve over the shank of the ophthalmoscope; it is provided with a snap catch (*H*) on the end to prevent the ophthalmoscope from falling out of the sleeve.

The holster snaps readily over the wearer's belt and may be worn conveniently, as illustrated in figure 2.

This holster is a product of the Peluse Specialties, 105 South Jefferson Street, Chicago.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

PHOTOGRAPHY IN OPHTHALMOLOGY

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The study of art, particularly of the paintings of the thirteenth and fourteenth centuries, shows that the artists' concepts of anatomy, from the present day standpoint, at least, were crude and impossible. Even today the most skilful portrait painter combines his interpretive insight with his manipulative skill. So it must necessarily follow that the old statement "Things are to the eye that sees them as to that eye they seem to be" is still true. It is one thing to have a desire to make a picture of beauty and another to get a faithful reproduction of the details. If one wishes scientific accuracy a photograph is necessary.

Photography has advanced so far that it needs no defense, but occasionally an explanation of how to use it tends to widen its field of application and make the results more presentable and more worth while. This paper is designed to draw attention to some of the practical rules of photography and the particular reasons for using different cameras in the special field of ophthalmology. It is a presentation of the methods I use.

When certain underlying basic principles are understood, all photography becomes comparatively easy and simply a matter of technical control while using the subject, manipulating the camera, adjusting the source of light, selecting the sensitive plate, developing it and, finally, printing from the negative.

I have found it easier to take a satisfactory photograph of the face or of the external portion of the eye when the patient is seated in a comfortable chair with the head placed against a white silk screen, which is not held taut but allowed to hang in loose folds. The illumination comes from two no. 2 Mazda photo-flood lamps in the ordinary Eastman Kodaflector, placed 3 feet (91.4 cm.) from the subject in a position to bring the lights and shadows with the desired contrast on the patient. Almost any camera may be used for this purpose, but it must be on a tripod, because the time of exposure is too great for ordinary steadiness of hand. I use the large Graflex camera, the National Graflex camera or the Leica camera. I prefer the large Graflex camera, because with it the subject is visualized up to the instant the picture is taken.

Most of my pictures of the external portion of the eye are made with the camera 3 feet from the patient. A Weston light meter is used to determine the size of the diaphragm opening and the time of exposure. The correct use of this instrument will avoid many disappointments caused by faulty exposure.

To focus the patient's face accurately the diaphragm is opened to its maximum size and then closed to 1/16 or 1/32, and when all is in readiness the plate is exposed for the predetermined proper length of time, which is with the former two-fifths second and with the latter one and one-half seconds, if a film with a 16 value, such as Verichrome, is used. Other types of film call for different times of exposure. In the ordinary case, speed is not essential for this kind of work, and therefore any of the standard double-coated films or plates can be used.

When one is working with a miniature camera, the position of the patient, the light and the camera are arranged in a manner similar to that for the larger apparatus. The built-in focusing mechanism enables one to adjust the camera to the selected distance. Great definition of depth is secured with the Leica instrument.

There are two ways of taking stereoscopic photographs with this camera. By one method the first picture is made in the usual manner; the camera is then moved a predetermined distance on the stereo slide bar, and the second picture is made under the identical conditions which pertained to the first. The other way is to use the stereoly attachment, by means of which the stereoscopic pictures are made with one exposure. With this method photographs are taken in a few minutes and supply an invaluable record of tumors, exophthalmos and deviations in the facial contour.

My clearest pictures of the external portion of the eye are made with the large Graflex camera, a 1/32 diaphragm opening for a one and one-half minute exposure of a Verichrome film being used. Because of the ease of handling, I prefer the film, but of course a plate can be used, and this gives ideal results.

A longer exposure is necessary to take a colored photograph. The light must be correct and the focus and exposure perfect, for it is essential that everything be accurate if one is to have clear pictures. More attention to detail is necessary when using color plates than when taking photographs in black and white. When a filter is used it should be placed before the photographing lens rather than over the source of light. This, I believe, is important.

When changing from one type of film to another it is necessary to remember the different time of exposure, and one should adjust the diaphragm accordingly. The proper exposure should always be calcu-

lated by using the light meter, for even after long experience I find that the complexion of the patient has much to do with the definition secured on the film.

For photographs of the lids, conjunctiva and anterior segment of the eye, I use one of three cameras, the Bausch and Lomb Stereocamera, the Zeiss Iris stereoscopic camera and the Drüner stereoscopic camera. Each has its field of use and its limitations.

The Bausch and Lomb Stereocamera is easy to operate. The patient's head is held in a rigid frame with a chin cup and a forehead rest. The light comes from two ordinary motor headlight bulbs projected through a lens system, which is an integral part of the camera, which in turn is mounted on a rigid base with adjusting screws. The part to be photographed is focused on the ground glass plate, and its position is noted through the focusing tube. The plate holder is placed in proper position, and the time of exposure is determined. The object is kept under control by observation through the focusing tube up to the time of exposure. There is no adjustable diaphragm with this camera, so that it is easy to get a clear stereoscopic picture by the exposure of ordinary orthochromatic plates for one twenty-fifth second. A filter is necessary when one is using the Dufay plates but not when using the Agfa color plates. For the latter a one-half second exposure gives the best results.

Infra-red plates should be used when the cornea is opaque. An exposure of one second gives surprisingly excellent pictures.

The Zeiss stereoscopic camera has a somewhat limited range of usefulness. It is well adapted to taking photographs of the eyelids, conjunctiva, eyeball and iris. It is a compact, excellent instrument with a built-in light, a tube for observation and a diaphragm control, but, unfortunately, it is not made for the present day color plates. The patient is held firmly against a forehead rest and a chin rest, and the plate is exposed in the usual way.

With the Drüner stereoscopic camera the object is focused on the ground glass plates, with control by observation through a telescopic focusing tube. The light is not a part of the instrument and must be supplied from an external source. The diaphragm opening is fixed, and the time cannot be regulated, so that to get a clear photograph it is necessary to have an intense illumination. I use a carbon arc lamp on an adjustable standard. The advantage of the instrument is that one can photograph all the parts of the eye back to the posterior portion of the capsule of the lens. Therefore, this is the camera of choice in photographing corneal changes, including endothelial deposits, altera-

tions of the iris and abnormalities of the lens. It is much more difficult to use than the other cameras, but no other gives such a wide range with considerable magnification of the object.

PHOTOGRAPHY OF MOVING OBJECTS

For the permanent record of defects of ocular motility, especially lesions of muscles, nothing serves as well as a clear moving picture. Changes can be portrayed just as well in the plain black and white film as in the color film. Kodachrome A film sensitized for photoflood lights gives beautiful colored pictures, and is both practical and inexpensive. The camera and the flood lights are placed so as to illuminate the object clearly and give the shadows their proper value. The size of the diaphragm opening is regulated by the reading on the photometer. It is not hard to take good moving pictures if the field is kept well lighted and in focus. The surgeon with extensive clinical opportunities and expert operative skill who wishes to record his observations should use the moving picture camera in daily practice.

PHOTOGRAPHS OF THE FUNDUS OCULI

The problems confronting the photographer of the fundus are by no means insuperable. If due attention is paid to the necessary details any one can make excellent pictures. Even at the risk of being elementary, it seems advisable to deal with this part of the technic step by step rather than by giving a general outline.

Preparation of the Patient.—The pupil must be dilated as widely as possible. This can be done with homatropine or eucatropine.

Adjustment of the Light.—The carbons of the arc lamp must be constantly adjusted. The direct current must be under complete control, 5 amperes and 110 volts being used. The carbon carrier must be placed so that the maximum brilliant illumination is secured. This is done by holding a card a few inches in front of the lens while moving the carrier to the site where the greatest efficiency results and locking it in that position. Many beginners experience difficulty by failing to heed this important recommendation.

Position of the Patient.—The patient's head is fitted into the forehead rest by changing the position of the chin cup by means of the adjusting screw which controls it and by raising or lowering the stool on which the patient sits. The head must be held rigid during the entire time of focusing and picture taking. I prefer to have an assistant keep it firmly in position.

Focus of the Light.—The band of light is partly focused on the side of the eye to be photographed. The other eye is fixed on the object held by the assistant or on the spot in a mechanical fixation device.

I prefer to use an assistant, for her hand can be moved by me as I desire to change the position of the eye. The camera light is then turned into the pupillary area and focused as a narrow band on the cornea, preferably just above the inferior edge of the pupil.

The position of the light and the angle at which it enters the pupillary space will depend on the portion of the fundus to be photographed, and an effort should be made to keep the light in the place where the intensification of lights and shadows give the clearest view. The time of exposure is previously determined by the ophthalmoscopic examination of the fundus. The plate is placed in the holder, and the slide removed, while the photographer continues his observations through the ocular. When all is in readiness the mirror is released and the shutter automatically opened. My best pictures of the average fundi are taken in one-half second on a double-coated orthochromatic plate. For pale fundi the time is reduced, and for dark ones it is increased.

Stereoscopic Pictures, Colored Pictures and Infra-Red Photography.—When a stereoscopic picture is desired, the base of the camera is displaced 44 mm., and the process is repeated. It is essential—in fact, absolutely necessary—that the photographer remember the precise spot where the light from the carbon arc was placed in the fundus view and bring the eye to that identical position. After everything is adjusted the plate is exposed in the same manner as for the preceding picture.

Colored pictures are taken on Agfa plates, and instead of lengthening the time of exposure I increase the illumination, using 7½ amperes and 110 volts, with direct current.

Infra-Red Photography.—For infra-red photography it is necessary to remove the fixed filter from the camera and put another one in its place. So far I have found little advantage in infra-red pictures of the fundus.

Colored fundus photographs are of increasing value, but as yet they cannot take the place of black and white photographs in showing details of the structure of blood vessels. They have an appeal to the general physician because he sees the fundus in a way similar to that to which he has become accustomed. The trained photographer also knows that the colored picture helps to locate some of the deeper retinal and choroidal lesions, particularly those which are overshadowed by a hazy retina. Colors are also desirable in papilledema, papillitis, retinal hemorrhages and exudates. My most beautiful color pictures are taken on Agfa plates without the interposition of a filter. Because of the construction of the fundus camera it is a little difficult to obtain any great degree of success with filters.

As the camera has no diaphragm control the exposure must be regulated by changes in the intensity of the illumination and in the time of exposure.

Stereoscopic photography of the fundus with the stereoscopic camera has not proved practical. To secure a good picture with the instrument it is necessary to have a dilatation of the pupil of from 7 to 8 mm., and the practicing ophthalmologist appreciates that it is impossible to get this in several conditions, notably, hypertensive states.

A camera is on the market for the photographic reproduction of the corneal surface by means of placedo disk reflections. The latest camera is for photographing the angle of the anterior chamber, but as yet this has not been perfected in mechanical arrangement, and the results leave much to be desired.

Comment.—Every clinician should take photographs; how many photographs he takes and under what conditions depend on his personal interest. Every ophthalmologist should be competent to read a photograph of the fundus. The day has passed when the ophthalmologist can go on the witness stand and say that he is unable to interpret a picture of the fundus, for this is a frank admission of his lack of knowledge, understanding and progressiveness.

SUMMARY

A satisfactory picture depends on the selection and manipulation of the camera made for the particular purpose, the correct angle and intensity of the illumination and the proper film. The smaller the diaphragm opening, other factors being equal, the greater the depth of the picture; so an attempt should always be made to observe this rule.

Until the photographer has become skilled he should entrust the development of the film and the printing of the picture to a trained technician, preferably a commercial photographer, who has by training and adaptability proved his worth as a cooperative agent.

In answer to many requests I include the list of the films I use:

For photographs of external conditions, the Eastman Verichrome film.

For photographs showing details of the lens and cornea, Hammer Ortho Plates Special Non-Halation, D. C.

For plain motion pictures, the Eastman panchromatic film.

For color motion pictures, the Eastman Kodachrome "A" film.

For infra-red photographs, Eastman infra-red plates.

For black and white pictures of the fundus, Hammer Ortho Plates. Special Non-Halation, D. C.

For colored pictures of the fundus and for colored pictures of the external part of the eye, Agfa color plates.

Since the early part of March, I have been using Kodachrome A in a film carrier which I had made for the fundus camera. The photographs are the best colors I have ever had.

Correspondence

OPHTHALMOLOGIC NOMENCLATURE

To the Editor:—I venture to call the attention of the ophthalmologic bodies to the desirability of appointing a committee on ophthalmologic nomenclature. Such a committee should go into the subject thoroughly and standardize and simplify many of the ophthalmologic terms which are at present used loosely or incorrectly.

To mention one term, let prism diopter be considered. As every one knows, the unit and the term were coined by Mr. Charles F. Prentice. Both have long been adopted by all the American manufacturers, who for several decades have been grinding their prisms and calculating all decentrations of lenses in terms of the prism diopter. Still many published articles continue to speak of prisms in degrees, and many a prescription is written for a prism of, say, 2 degrees. It is safe to state that the prism used in the test was a prism of 2 prism diopters, and certainly the prism in the prescription will be a prism of 2 prism diopters, since prisms are no longer ground according to the degree system.

In the recent work on ocular muscles by Dr. Peters one finds prisms spoken of in terms of centrads. The centrad proposed by Dr. Dennett was never adopted in this country, and prisms are not ground on the centrad system. I believe that Dr. Peters meant prism diopters when he wrote centrads; yet the centrad and the prism diopter are different units. The former is based on an angular measurement; the latter, on a linear measurement. It is true that the actual effective difference between prisms made according to the three systems of degree, centrad and prism diopter is slight. Still, it is inaccurate to mix the terms indiscriminately.

The general adoption of a correct terminology for prisms would eliminate a frequent source of confusion in discussions of the measurements and treatment of squint. For instance, when in the literature a condition is reported as a squint of, say, 15 degrees, one frequently does not know whether that means 15 degrees of actual angular deviation as measured on a perimeter, a method which Dr. Peters favors, or 15 prism diopters as measured by the cover test with loose prisms, as practiced by Dr. White. If the word "degree" is never used to denote prism values, any reference to degrees would mean only one thing—angular deviation as measured on a perimeter or a similar instrument.

I believe the principal reason for the lag in the use of the term prism diopter is the awkwardness of the term as compared with the terms degree and centrad. Various attempts have been made to simplify the term prism diopter to prismopter, prisopter and similar substitutes. But these terms are still too clumsy. A committee on nomenclature could agree on a suitable term and have it generally adopted. Personally, I have frequently used the word "delta," which is the name of the Greek letter Δ, at present used as the symbol for prism diopter. This seems a suitable term and is as short and euphonious as "degree" or "centrad." Thus one would speak of a 1 delta prism, a 5 delta prism, etc.

Another desirable matter for such a committee to take up would be the advisability of describing the position of the prism by its apex rather than by its base. The image seen through a prism is in the direction of the apex, and a prism both for measuring and for relieving heterophoria is placed with its apex in the same direction as the deviation of the eye. It is much easier to correlate the position of the apex of the prism with the ocular deviations than it is to correlate that of the base, the latter correlation requiring inversion and some mental gymnastics. This is a waste of mental energy and hampers rapid and effective thinking.

The foregoing are only a few suggestions. There are many more terms which could be simplified and standardized by such a committee in the interests of clearer and more accurate thinking and writing.

JOSEPH I. PASCAL, M.D., New York.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Examination by the American Board of Ophthalmology.—The American Board of Ophthalmology announces that, in addition to the examination to be held in Philadelphia on June 7, 1937, another examination will be conducted in Chicago on October 9. All applications and case reports, in duplicate, must be filed at least sixty days before the date of examination. Further information may be secured from the secretary, Dr. John Green, 3720 Washington Boulevard, St. Louis.

SOCIETY NEWS

American Academy of Ophthalmology and Otolaryngology.—Announcement has been made by the American Academy of Ophthalmology and Otolaryngology that the meeting place of the 1937 convention has been changed from Detroit to the Palmer House, Chicago. The convention will be held from October 10 to 15.

French Ophthalmologic Society.—The One Hundred and Fiftieth Congress of the Société Française d'Ophthalmologie will take place in Paris, at the Faculté de Médecine, on June 28, 29 and 30.

Philadelphia County Medical Society, Eye Section.—The following program was presented at the scientific meeting of the Eye Section of the Philadelphia County Medical Society, held on March 4:

Presentation of clinical cases from the Wills Hospital, by Dr. Henry Irwin.

"Albinism from the Standpoint of Pigment Defects," by Dr. Howard Rome.

Discussion of albinism, by Dr. P. De Long.

"Further Studies in Dark Adaptation as a Clinical Test," by Dr. Jacob B. Feldman.

Discussion of dark adaptation, by Dr. Edmund B. Spaeth.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Aqueous Humor

THE AMOUNT OF OXALIC ACID IN THE AQUEOUS. O. BARATTA, Arch. di ottal. **43**: 44 (Jan.) 1936.

Baratta followed Maugeri's method, employing 5 cc. portions of aqueous from the eye of the horse, ass, ox, calf, goat and sheep. For the smaller animals, the smaller amounts of aqueous obtainable were diluted with water to 5 cc. Eight to twelve samples were run for each species of animal. The amounts of oxalic acid found, in milligrams per hundred centimeters, were 7.8 in the horse, 7.2 in the ass, 9 in the sheep, 7.9 in the calf, 8.4 in the goat and 8.61 in the ox. The presence of oxalic acid in the human aqueous was determined, but the amount was not estimated.

S. R. GIFFORD.

Biochemistry

SULPHUR METABOLISM IN SENILE CATARACT. M. C. BOURNE and D. A. CAMPBELL, Brit. J. Ophth. **20**: 684 (Dec.) 1936.

Bourne and Campbell state: "The results of this investigation do not reveal any difference in the urinary sulphur distribution in the cataractous patients as compared with the control cases. Although the individual variations are great, the results are in all cases similar, and in general follow the classical distribution of urinary sulphur first described by Folin (1905). That is, the largest fraction of the total sulphur is excreted as inorganic sulphate which varies with the amount of protein ingested; while the ethereal sulphate and neutral sulphur fractions remain more or less constant and independent of the total sulphur excretion. In none of the patients with senile cataract is any deviation from the normal to be observed in the behavior of any fraction. Therefore the results lend no support to the hypothesis that a disorder of sulphur metabolism might be the basis of the pathological changes in the lens in senile cataract; but the experiments are not sufficiently extensive or conclusive to rule out the suggestion completely. A detailed study of the neutral sulphur fraction, the composition of which even in normal individuals is not completely understood, might show the presence of some specific compound associated with senile cataract. The fact that no increased excretion of the ethereal sulfates was found in any of the cataractous patients in our series makes it unlikely that senile cataract is due to a toxic process. Blood glutathione estimations were done on a number of these patients; the results, which have already been published (Campbell, 1936), showed a normal blood glutathione in every case."

W. ZENTMAYER.

EFFECT OF ALPHA-DINITROPHENOL ON THE OXIDATION-REDUCTION POTENTIAL OF THE AQUEOUS AND THE LENS. J. NORDMANN and P. REISS, Compt. rend. Soc. de biol. **123:** 233, 1936.

Attempts to produce lenticular opacities in the rabbit and the rat with dinitrophenol were unsuccessful. In normal rabbits the potential of the aqueous and the lens was found to be plus 143 millivolts and plus 116 millivolts, respectively. However, after the fourth injection of dinitrophenol a marked elevation of the potential was maintained, averaging for the aqueous and the lens plus 168 millivolts and plus 159 millivolts, respectively, which persisted after cessation of the treatment.

J. E. LEBENSOHN.

Conjunctiva

THE EYE AND GENERAL INFECTIONS: CONJUNCTIVITIS AND GONOCOCCIC ARTHRITIS. E. PUSCARIU and D. LÂZÂRESCU, Ann. d'ocul. **173:** 893 (Nov.) 1936.

Infection in the eye is met with in two forms: first, the exogenous form (the more frequent) and, second, the endogenous form, which is responsible for metastatic ophthalmic lesions. The pathogenic agents may penetrate into the organism after entering the eye. Many references are given in the literature to observations on septicemia, meningitis, abscesses or arthritis in which the eye served as the point of entrance to such pathogenic agents as streptococci, staphylococci and pneumococci.

From the statistics of the ophthalmologic clinic at Jassy the following figures are taken: From 1919 to 1935, among 37,960 patients there were found 461 with purulent ophthalmia (1.02 per cent). When the total number of cases of conjunctivitis is considered, the percentage is 4.09 per cent. The patients were examined, as a rule, from the seventh to the thirtieth day after the beginning of the ophthalmia, but generally in the last week. Among the 461 patients with gonococcic conjunctivitis, there were 3 in whom the condition was complicated with arthritis—2 babies and the mother of one of these. Observations on these 3 patients are given in detail. One of the patients had polyarthritis; in the other 2 the localization was mono-articular.

After treatment with antigonococcus vaccine, intramuscular injections of milk in the babies, and infra-red rays, the articular lesions were considered cured. An extensive bibliography is given.

S. H. MCKEE.

KERATITIS PUNCTATA SUPERFICIALIS AND SWIMMING POOL CONJUNCTIVITIS W. P. LING, Chinese M. J. **50:** 1381 (Oct.) 1936.

This article consists in the report of fifty-three cases of keratitis punctata superficialis in Chinese patients, in nearly half of whom the condition was associated with swimming pool conjunctivitis, which was particularly prevalent in Nanking during the summer and autumn of 1934. There appeared to be a relationship between the keratitis and the swimming pool conjunctivitis, although the former could occur independently of the latter. It is possible that both these conditions had the same etiology, which, of course, is still unknown.

The simultaneous occurrence of these two conditions in the same patient is new to the author and does not seem to have been mentioned in the literature. One must bear in mind that swimming pool conjunctivitis is not confined to swimming pools. As the infection is probably of genito-urinary origin it can naturally occur under any circumstances and may be transmitted from one person to another at any time. Accordingly, some authors have suggested the name "adult inclusion conjunctivitis," or "inclusion blennorrhea of the adult."

S. H. McKee.

Congenital Anomalies

A Case of Dissociated Albinism. J. Bollack and Delthil, Bull. Soc. d'opht. de Paris, October 1935, p. 623.

A woman of 29 years showed on oblique illumination of the eyes a chestnut-brown iris with a red coloration of the pupil. Both the iris and the sclera were especially transparent to reflected light. Horizontal nystagmus was present in all aspects. The entire fundus was pale, as though the retina rested on the detached network of the choroid. In the intervascular spaces of the choroid there was no evidence of pigmentation. There was no differentiation of the macula, and no pigment was present in this area. The fovea was indistinct, and only tortuous vessels could be seen. The normal reflex of the retina was entirely absent, particularly at the fovea. There was no pigment in the posterior layer of the iris, but the stroma had its normal brown pigment, and the entire uvea was translucent. The contour and reaction of the pupil were normal. The cornea, lens and vitreous were normal.

L. L. Mayer.

Cornea and Sclera

Contact Glass as a Therapeutic Agent in Corneal Ulcers. D. M. Rolett, Am. J. Ophth. **19:** 888 (Oct.) 1936.

Rolett reports two cases of corneal ulcer, in one of which the ulcer was associated with hypopyon and in the second of which it was due to neuroparalytic keratitis and lagophthalmos. In the first case atropine and strong protein silver were used in the contact glass, and in the second case Ringer's solution was used. Both patients were discharged as cured in ten and nine days, respectively. Rolett calls attention to the simplicity of this treatment and its brilliant results.

W. S. Reese.

Transplantation of Cornea. J. W. T. Thomas, Irish J. M. Sc., July 1936, p. 289.

Thomas has done thirty-six consecutive operations for corneal transplantation, twenty-four of which were performed on twenty-one suitable eyes. About 80 per cent of the patients were benefited by the operation; in 28 per cent the grafts were clear, and vision was 6/36 or better. Seventy per cent of the operations were successful, and 25 per cent resulted in clear grafts. The author quotes the results of Elschnig,

Filatov and Castroviejo, from which it is seen that the operation for corneal transplantation is one that should be included in the list of operative procedures which can be legitimately employed in suitable cases. The study of the operation is yet far from completed. Attention to detail is an essential requirement, and minute experimental departures from the routine procedure should be tried singly and not collectively, so that a logical inference may be drawn from the result. The outlook in the future for patients more or less blind from corneal opacities is decidedly encouraging, and in suitable cases there is even now a 70 per cent chance of improvement and a 25 per cent chance of obtaining good useful vision. The future will hold some chance of improvement in many of the cases in which the condition is, for various reasons, classified as unfavorable.

J. A. M. A. (W. ZENTMAYER).

KERATITIS ULCEROSA AND NONULCEROSA PRODUCED BY DIPLOBACILLUS PETIT. P. S. SOUDAKOFF, Chinese M. J. **50:** 1415 (Oct.) 1936.

This article consists in the report of three cases of corneal ulcer in which a relatively rare micro-organism was found to be the cause. The diplobacillus of Petit was first described by him in 1898, and was studied serologically by Chaine in 1914, who proved that it was not an evolutionary form of the diplobacillus of Morax and Axenfeld but a variety of the same family.

Keratitis produced by the diplobacillus of Petit either takes a mild course, the patient showing multiple nonulcerating infiltrates of about three weeks' duration, or appears as crescent-shaped relatively benign ulcers. The condition generally reacts satisfactorily to conservative treatment.

S. H. MCKEE.

Experimental Pathology

SOME REMARKS ON OEDEMA. J. A. VAN HEUVEN, Brit. J. Ophth. **20:** 631 (Nov.) 1936.

After recounting the effects of instilling oleum sinapsis into the conjunctival cul-de-sac van Heuven comments on the chemosis which results. It might be thought that liquids reach the tissues. However, it appears to be impossible to recover liquids by means of puncture, incision or squeezing. The use of vital staining was tried, a 1 : 800,000,000 solution of fluorescein and a 1 : 1,000,000,000 solution of acriflavine hydrochloride being employed. The examinations were made with the biomicroscope under arc light illumination through quartz glass and Uvet filters. The injections were given intravenously after instilling oleum sinapsis. From these experiments it appears that in inflammations the normal circulation of the blood is disturbed, as well as that of the lymph. In the lymph this change is opposite to that of the blood. Without vital staining there is an increase in the efferent current, whereas with vital staining there is an increase in the afferent current. In inflammation there is an increase in the permeability of the vessels, and the liquid leaves the vessels and is incapable of reentering them. It is through the lymph vessels that the liquid leaves the tissues; the

capacity of the lymph vessels is insufficient to absorb the total amount, and the chemical picture of chemosis results. Many other interesting questions in connection with these experiments are discussed.

W. ZENTMAYER.

General

A CRITICAL SUMMARY OF SURGICAL EXPERIENCES IN 1934. E. HILL and R. H. COURTNEY, Am. J. Ophth. **19:** 773 (Sept.) 1936.

This article does not lend itself to abstracting. It is a report of the experiences of the ophthalmic staff of the Medical College of Virginia during 1934 in both private and clinical practice, based on 239 operations.

W. S. REESE.

OPHTHALMOLOGY AND RESEARCH. JOHN PARSONS, Brit. J. Ophth. **21:** 1 (Jan.) 1937.

This is an address delivered before the American Academy of Ophthalmology and Otolaryngology. It does not well lend itself to abstracting. The introductory remarks are on vision, and it is pointed out that the study of normal vision demands no small knowledge of physics, chemistry, physiology, neurology and psychology.

The present status of the theory of the function of the intra-ocular fluid is discussed. Much of the evidence to show that osmotic interchange is more important than was once thought depends on minute physical and clinical measurements which are perilously near the borderline of error. Parsons states that he is always rather suspicious of chemical weighing carried to four decimal places!

The newer view concerning the nature of the vitreous is discussed, and the chemistry of the lens and cataract are considered. More recent studies of the circulation of the fluids and the cause of glaucoma are reviewed. Parsons has an open mind on most of these subjects. The paper will well repay reading.

W. ZENTMAYER.

General Diseases

CLINICAL ASPECTS OF OCULAR TUBERCULOSIS. F. MEHLMACK, Arch. f. Augenh. **110:** 39, 1936.

Three hundred and thirty-five cases of ocular tuberculosis are reported from Hertel's clinic in Leipzig. The largest percentage of patients were between 20 and 50 years of age. The majority of the patients between the twentieth and thirtieth decades were women. The uveal tract was the seat of the disease in 80 per cent, and the patients apparently suffered chiefly in two periods of life, either early in youth or later (in the case of women, at the time of the menopause). In practically all the cases of uveal tuberculosis primary lesions were found in the chest. The tuberculin reaction was positive in practically all the cases in which clinical and roentgen examination revealed inactive tuberculosis, but in a number of the very young patients and in some of the women during the menopause the reaction was negative in spite of definite evidence of active generalized tuberculosis, such as involvement of the lymph glands with or without pulmonary involvement.

Mehlmack does not believe that the exudative form of uveal tuberculosis is limited to any one age group. A general résumé of the treatment as carried out in Hertel's clinic is given. Sixty-five per cent of the patients were cured.

F. H. ADLER.

Glaucoma

CHRONIC GLAUCOMA WITH LOWERED ARTERIAL TENSION, TREATED WITH A CALICUM-MAGNESIUM PREPARATION. JEAN GALLOIS, Bull. Soc. d'opht. de Paris, December 1935, p. 801.

A patient of 61 years had bilateral chronic glaucoma with the usual symptoms. Pilocarpine controlled the tension, but the halos persisted, as did an arterial pulsation. Later, visual acuity became reduced, and the question of an operation arose. Resort to injections of a calcium-magnesium preparation as recommended by P. Viard resulted in complete control of the visual acuity, visual fields and tension. A chart showing the tension is included. The drug is used in the following combinations: calcium chloride, 0.75 Gm.; magnesium thiosulfate, 0.8 Gm., and water, to make 10 cc.

L. L. MAYER.

ANTERIOR CHAMBER PUNCTURES: AN AID IN THE DIAGNOSIS OF GLAUCOMA. P. C. KRONFELD and LIN CHING-K'UEI, Chinese M. J. **50:** 1323 (Oct.) 1936.

The writers describe the technic of, and past experiences with, puncture of the anterior chamber in the diagnosis of primary glaucoma in the early stages. The qualifications for Chinese control cases are discussed, and the results obtained in the tests on the controls are given.

In twelve patients with primary glaucoma in the early stages or with signs suggestive of this condition, puncture of the anterior chamber was performed; this resulted in a typical and more pronounced hypertensive reaction than that obtained in the controls, in ten of the twelve cases.

Puncture of the anterior chamber is recommended as a safe and expedient provocative test for the detection of early or latent primary glaucoma. A bibliography is given.

S. H. MCKEE.

Hygiene, Sociology, Education and History

OCCUPATIONAL ABILITY OF INDIVIDUALS OPERATED ON FOR CONGENITAL CATARACTS. K. VOM HOFE, Arch. f. Augenh. **110:** 35, 1936.

Of twenty-one patients with bilateral congenital cataracts who had been operated on, ten had good visual acuity. Nine had sufficient visual acuity to pursue their usual occupation. In only two was the visual acuity insufficient for the patients to learn a trade. In the vast majority vom Hofe found the visual acuity sufficient for the following employments: locksmith, carpenter, merchant, farmer, messenger, servant, student, clergyman.

F. H. ADLER.

A Short History of Old Chinese Ophthalmology. Lee Tao, Chinese M. J. **50:** 1513 (Oct.) 1936.

A brief historical sketch of Chinese ophthalmology is given, and the suggestion is made that a great part of Chinese ophthalmology is based on Indian medicine. The knowledge of the anatomy of the eye was scanty, and diseases of the eye in most cases were ascribed to internal diseases.

Many diseases of the eye are mentioned in ancient Chinese literature, and the different forms of treatment given for most of them may be considered to be more or less useless.

S. H. McKee.

Injuries

The Minor Sequelae of Eye Contusions. M. Davidson, Am. J. Ophth. **19:** 757 (Sept.) 1936.

From a study of thirty-four contusions of the eye and an analysis of the sequelae Davidson found a prevailing concomitance of from two to six lesions, obviously anatomically related to each other and stretching from the root of the iris along the equator of the lens, the zonule, the ciliary body, the ora serrata and the vitreous which justifies the notion of the syndrome of trauma of the anterior segment described by Henri Frenkel. This is of medicolegal importance in the presence of a lesion of the anterior segment. Davidson discusses the different theories of contusion and some of the problems in its mechanism that still require study.

W. S. Reese.

Accidental Freezing of the Eye by Sulphur Dioxide. C. P. Clark, Am. J. Ophth. **19:** 881 (Oct.) 1936.

Clark gives the following summary:

"1. The history and clinical phenomena are related of two eyes that received serious impairment from a sudden jet of liquid sulphur dioxide.

"2. The result of this accident was sudden and intense freezing of the ocular structures.

"3. There was a central facet in each injured cornea.

"4. The following treatment is suggested: Wash the eye at once with cold water (ice water would be preferable) and follow with the application of iced compresses. Local anesthetic and cycloplegic drugs should be employed in sufficient amount to maintain their therapeutic effect. An antiseptic ointment should be applied between the eyelids. Emphasis is given to the prompt application of cold water to the injured eye because if this is delayed much greater tissue damage will result."

W. S. Reese.

Immediate Damage to the Iris and Lens from an Ammonia Burn. W. Kiss, Arch. f. Augenh. **110:** 98, 1936.

An eye in which a 10 per cent solution of ammonia had been splashed presented the following appearance when first seen in the clinic:

The conjunctiva was gray and opaque, with many hemorrhages scattered throughout. The corneal epithelium had entirely disappeared, and the eye was insensitive. The anterior chamber was deep and gave a Tyndall effect. The iris was semidilated and did not react. At the upper margin the stroma was pushed forward, and the pigment epithelium, which had become separated from the stroma, hung down in the form of a curtain, in folds. The lens showed some indefinite clouding.

Ten months after the injury the eye was enucleated for cosmetic reasons. The layers of the iris were found to be separated by a transudate, which Kiss believes came from the vessels of the iris when the strong solution of ammonia diffused through into the anterior chamber.

F. H. ADLER.

INFLUENCE OF RED LIGHT ON THE EYES OF PHOTOGRAPHY WORKERS. D. M. NATANSON and D. P. VINOGRADOW, Sovet. vestnik oftal. **8:** 852, 1936.

Seventeen photography workers were examined during the working hours (from four to eight hours). Anomaly of refraction was observed in fifteen; the color sense was normal in all, and accommodative asthenopia was observed in four. The light sense, which was examined carefully, was found to be reduced to one fourth of normal. The common pathologic change in all the workers was chronic conjunctivitis, which evidently was caused by prolonged work under poor lighting and by the action of red and infra-red rays on the conjunctiva.

O. SITCHEVSKA.

Lacrimal Apparatus

RATIONAL THERAPY OF PATIENTS WITH CATARRHAL CONDITION OF THE LACRIMAL APPARATUS. I. V. SERGEEV, Sovet. vestnik oftal. **9:** 222, 1936.

Since Sergeev considers stenosis of the lacrimal punctum one of the chief causes of epiphora, he uses the following surgical method for correction of this condition: The lower punctum is dilated with a dilator; a probe, size 1, is introduced up to the nasal bone, the lumen being dilated as much as possible by pulling the lower lid down and out. Then a discission knife is introduced with its cutting surface upward, and the medial wall of the canaliculus is incised for from 2 to 3 mm. The probe and knife are removed; the lateral lip of the wound is grasped with forceps, and a triangular piece is cut out with scissors so that a wide opening of the punctum is made. A fibrinous blood clot forms under the dressing, which is removed in two or three days.

One hundred and twenty-nine patients were treated by this method. Seventy, or 55 per cent, were cured; in fifty-three, or 41 per cent, the epiphora was partially improved, and in four there was no change in the condition. The visits to the dispensary were reduced to one-fourth. The majority of the patients were observed for about eight months.

O. SITCHEVSKA.

Lens

ANTERIOR LENTICONUS. E. C. MOULTON, J. A. M. A. **107:** 1809 (Nov. 28) 1936.

According to Moulton, the case here described is the eleventh of the cases of the true type of anterior lenticonus reported. The patient was a man aged 33. Since the age of 17 he had been aware of poor vision. Vision in a darkened room was 20/100 (6/30) in the right eye; with a + 1.00 sphere it was 20/70 (6/21). In the left eye it was 20/70 (6/21); with a +1.00 sphere it was 20/50 (6/15).

With the ophthalmoscope the characteristic "oil drop" was observed in the center of the pupil. With the pupil under the influence of atropine the fundus was plainly seen through the peripheral emmetropic lens substance. Through the apex of the cone the fundus was plainly seen with a —20.00 lens. No changes in the fundus were seen.

Iridectomy was performed in the lower nasal quadrant of the iris of the left eye. The resulting vision was 20/50 (6/15) and was not improved by a glass.

With the biomicroscope two zones of disjunction were seen to be involved, the anterior band of the lens and the anterior band of disjunction. These two bands were seen to bend forward over the clear underlying contacts of the conus, without alteration in their parallelism or change in the thickness of either zone. All the other zones were normal. On the apex of each zone and situated just beneath the intact anterior capsule were a few very minute and delicate brown granular opacities.

In the three cases of this condition reported to date in which the eye was examined with the slit lamp the disorder must have been the acquired form of anterior lenticonus, and in this case, as in the other two, it is a question rather of lentiglobus than of lenticonus.

W. ZENTMAYER.

INTRACAPSULAR CATARACT EXTRACTION. O. M. DUTHIE, Brit. J. Ophth. **21:** 16 (Jan.) 1937.

Duthie employs the technic of Sinclair modified only in minor details. Retrobulbar injection of an anesthetic is given twenty minutes before operation. An injection is also made into the lids, and subcutaneous injections are given in order that retraction sutures, which are used in place of a speculum, may be inserted painlessly. A large corneal section with a small conjunctival flap is made. The capsule of the lens is grasped as near the level of the lower pupillary margin as possible. Where the vitreous tends to bulge after extraction of the lens, gentle pressure is applied to the globe, the lids being closed, for one to two minutes. A peripheral iridectomy is preferred.

The author gives the following summary:

"1. The present paper deals with a series of 100 consecutive intracapsular cataract extraction operations.

"2. The results show a final visual acuity, in healthy eyes, of 77 per cent, 6/6 or better, and 19 per cent, 6/9-6/12.

"3. The most common single complication is hyphaemia—28 per cent—for which no suitable method of prevention is suggested.

"4. Iris prolapse occurred in 8 per cent and vitreous prolapse in 6 per cent of cases; suggestions are made with a view to reducing this incidence and at the same time making peripheral iridectomy a safer procedure.

"5. Minor points in local anesthesia are suggested.

"6. Superiority of the operation over other methods is claimed, particularly in immature cases."

W. ZENTMAYER.

HEREDITARY LUXATION OF THE LENS AND THE SYNDROME OF MARFAN. DELORD and VIALLEFONT, Bull. Soc. d'opht. de Paris, January 1936, p. 44.

A father aged 41 and his son aged 15 years showed various stigmas of the syndrome of Marfan. Both had bilateral luxation of the lens. Scoliosis, arachnodactylia, hyperactive reflexes, absence of secondary sexual characteristics, infantile testicles, a basal metabolism lowered 25 per cent and a blood calcium content of 0.13 per cent were found in the son, but the father was normal in all these respects. Delord and Viallefont discuss the various theories of the etiology of the condition. Excellent photographs of the son are shown in the article.

L. L. MAYER.

BIOMICROSCOPY IN TWO CASES OF DYSTROPHIC MYOTONIC CATARACT. G. VON GROLMAN, Arch. de oftal. de Buenos Aires **10:** 707 (Sept.-Oct.) 1935.

This is the report of a biomicroscopic examination of cataract in a sister and a brother of 31 and 34 years with dystrophic myotony. In the sister, who had abundant spherules of shagreen on the anterior capsule and a diffuse opacity of the cortical layers, the light reaching the superficial layers of the adult nucleus, the anterior suture wheel, divided into eight spokes, was quite clear; the diffuse opacities were uniformly distributed in a superimposed layer with bright reflection of light in the central and equatorial regions. The brother showed in the right eye a normal anterior capsule; the cortical opacity was uniform with spots of denser opacity in the form of facets, and there were white spots of a dull aspect in the equatorial zone. The anterior suture of the fetal nucleus was visible, and the fetal nucleus was more opaque than the adult; on the anterior Y bright points of a red, green or blue color according to the incidence of the light could be seen (Vogt's points). In the left eye of the same patient the anterior capsule was normal, with some spherules of shagreen; there was a diffuse dustlike cortical opacity arranged in layers and as a dense pointlike formation; direct illumination showed clearly that the fetal nucleus was much more opaque than the adult, and the anterior suture was clearly visible.

C. E. FINLAY.

THE AMOUNTS OF ACID-SOLUBLE ORGANIC PHOSPHATE IN NORMAL AND CATARACTOUS LENSES. H. K. MÜLLER, Arch. f. Augenh. **109:** 497, 1936.

Müller has proved that in the lens cevitamic acid is formed from sugar according to the following equation—sugar + oxidized gluta-

thione = cevitamic acid + reduced glutathione. On the basis of other work he believes that there is a close relationship between vitamin C and the amount of carbohydrate-phosphatide present in the lens, and the present work was undertaken to disclose this relationship.

The phosphatide content of young and old ox lenses was determined by the method of Fiske and Subbarow (*J. Biol. Chem.* **66:** 375, 1925), and it was found that young lenses contain more acid-soluble organic phosphate and more vitamin C than old lenses. Similar determinations were made on cataractous lenses from oxen, and it was found that less acid-soluble organic phosphate was present than in the normal lens.

Müller concludes that in the development of a cataract the lens not only loses vitamin C but also its acid-soluble organic phosphorus. On the other hand, the inorganic phosphate is usually not reduced in amount, and frequently more is found than in the normal lens, which corresponds well with the increase in calcium known to occur in cataractous lenses.

F. H. Adler.

Lids

Removal of the Tarsal Part of the Orbicularis Muscle of the Upper Lid for Trachomatous Entropion and Trichiasis. A. Busacca, Ztschr. f. Augenh. **88:** 100 (Jan.) 1936.

Busacca believes that the entropion and trichiasis complicating trachoma are due chiefly to hypertrophy of the marginal part of the orbicularis muscle, the hypertrophy coming from the spasm of the lid caused by the primary keratitis.

The removal of the muscle bundle is done through a single incision in the skin 0.5 cm. from the margin of the lid. The tarsus is thinned down to leave a step at the upper margin where the levator attaches. The lower edge of the skin is stitched to this step with five or six sutures. The upper edge of the skin is left free to form the normal fold of the lid.

The operation accomplishes the removal of hypertrophic muscle and holds the margin of the lid and the lashes away from the cornea. Several hundred patients have been treated by this method in the last four years, and no cases of recurrence have been observed by Busacca. Explanatory drawings accompany the article.

H. Gifford Jr.

Methods of Examination

The Clinical Value of Tryptophane-Reaction. J. A. van Heuven, Brit. J. Ophth. **20:** 635 (Nov.) 1936.

As a result of experimental study van Heuven concludes that the tryptophane reaction has been proved to be not a specific test of tuberculosis but a sensitive indication of increased permeability of the capillaries. An advantage of the reaction is that it allows one to demonstrate increased permeability by means of a substance normally existing in the circulation.

W. Zentmayer.

EXAMINATION OF THE EYE BY MEANS OF THE SODIUM LAMP. G. KLEEFELD, Bull. Soc. d'opht. de Paris, December 1935, p. 788.

A review of the history of the use of the sodium lamp, with some observations on its clinical application, is given. Kleefeld concludes that the sodium lamp presents a certain interest for the ophthalmologist. Its use in the study of the palpebral vessels and the conjunctiva and in focal illumination is of value. The lacrimal papilla is viewed with great ease, and atresias are brought out. Opacities of the lens and vitreous are more readily studied than with ordinary illumination. Because of the suppression of the red color, the fundus is more precisely studied. Localization of hemorrhages, especially preretinal hemorrhages, is easily made. Skiascopy is rendered more exact, and more remarkable contrast is noted in photography of the anterior segment of the eye than with other sources of illumination.

L. L. MAYER.

Neurology

RELATION BETWEEN DISEASE OF THE TRIGEMINAL NERVE AND INTRA-OCULAR OPERATIONS: REPORT OF A CASE. W. GILBERT, Klin. Monatsbl. f. Augenh. **97:** 70 (July) 1936.

Gilbert discusses the relations between disturbances in the area of the trigeminal nerve and intra-ocular diseases, with special reference to glaucoma and the divers types of ocular herpes, and also neuralgia. He reports the case of a woman aged 57 to show the serious effect of neuralgia of the first branch of the trigeminal nerve, which developed during recovery after extraction of a senile cataract. The patient had no ocular or other diseases at any time except neuralgia in the right side of the head for many years. Purulent iritis set in three days after the operation, while the corneal flap remained intact. Operation for secondary cataract was performed with De Wecker's scissors seven months later. A hemorrhage in the anterior chamber was observed three days later, and a number of hemorrhages from the temporal pillar of the coloboma of the iris occurred two or three times a week for about four months. These hemorrhages resembled those described by Gilbert in his paper on herpes. Secondary glaucoma, associated with severe attacks of neuralgia, necessitated enucleation. Histologic examination revealed necrosis of the upper portion of the iris and perineuritic inflammation of some ciliary nerves; the choroid and retina were intact. Gilbert thinks that herpes virus entered the trigeminal nerve endogenously, causing chronic inflammation in its branches up to the ciliary nerves. This process caused the neuralgia without involvement of the eye. The operation produced chronic neuritis in the ciliary nerves. Gilbert concludes that extraction of cataract should not be performed prior to six months after the last attack of neuralgia. It is advisable to perform preliminary iridectomy or avoid iridectomy altogether.

K. L. STOLL.

Ocular Muscles

PARALYSIS OF DIVERGENCE OF FUNCTIONAL ORIGIN. C. P. CLARK, Am. J. Ophth. **19:** 789 (Sept.) 1936.

Clark reports the case of a 26 year old white woman who under stress of economic and family troubles began to suffer from aching of both eyes and then diplopia. There was concomitant convergent strabismus, the deviation at 6 meters varying from 12 to 25 degrees. Fixation alternated between the two eyes, and the field of rotation for each eye was normal when the fellow eye was occluded. Binocular single vision was present when the test object was brought to within 60 or 70 cm. of the face. The near point of convergence was 6 cm. The condition eventually cleared up almost entirely. The Wassermann test and physical examinations gave negative results.

W. S. REESE.

A CASE OF CONVERGENCE SPASM. P. BAILLIART, Bull. Soc. d'opht. de Paris, February 1936, p. 81.

A girl aged 8 years had complained of double vision for eight days. The mother reported that the child had had such an attack twice previously, with accompanying convergent strabismus and apparent diplopia. The first attack was at the age of 2 years and lasted eight days; the second, which occurred after a year, was of short duration. These two attacks were thought to be due to eating chocolate. In the present attack the strabismus was 40 degrees. A lens to correct a refractive error of 1 diopter brought the visual acuity of the right eye to 10/10 and that of the left to 9/10. When the child put on her glasses (a + 1.0 diopter sphere) the convergence disappeared. Also, if her attention was distracted or she became greatly interested in something, no evidence of crossing was noted. Bailliart injected a 2 per cent solution of procaine hydrochloride into the right internal rectus muscle; this caused complete disappearance of the squint. Other facts of the history were unimportant. Physical examination gave entirely negative results. This included a complete neurologic examination. Both parents were examined. Bailliart thinks that the condition was spasm of convergence, which has been rarely recorded in the literature, as shown by the reports. In some cases the condition was noted after encephalitis. No etiologic factor was found in the described case.

L. L. MAYER.

Operations

CYCLECTOMY. L. VON BLASKOVICS, Ztschr. f. Augenh. **88:** 75 (Jan.) 1936.

A rather simple method for removal of a piece of ciliary body through a meridional incision is described. The horse hair sutures are placed through the edge of the sclera before it is cut completely through. After completion of the scleral incision the ciliary body is partly

detached with a cyclodialysis spatula, pulled into the wound and cut off with scissors. The sutures are carried into the conjunctiva so that its closure does not fall opposite the scleral wound.

Five cases are reported in which part of the ciliary body was removed. No serious complications developed that could be attributed to the operation. In one patient 23 years of age who had sustained a trauma of the eye in whom good vision was retained, there was no receding of the near point, and accommodation was undisturbed following cyclectomy.

Blaskovics feels that this is the operation to be chosen in removing nonmagnetic foreign bodies and in cases of rupture of the sclera with prolapse of uveal tissue. If there is no uveal tissue left incarcerated in the wound, he feels that there is no more danger of sympathetic ophthalmia occurring after this operation than following cyclodialysis.

H. GIFFORD JR.

Orbit, Eyeball and Accessory Sinuses

SYNDROME OF NASAL NERVE FROM BACILLARY TOXEMIA. C. CHARLIN, Ann. d'ocul. **173**: 25 (Jan.) 1936.

Charlin states that he knows well the tuberculous toxemia with loss of weight, asthenia, loss of color and sometimes vesper fever which occurs without clinically observable pulmonary lesions. Fluoroscopy and better still roentgenography show thoracic pulmonary shadows, especially at the hilus. The condition is generally found among young adults, especially females. In many cases with rest and tuberculin therapy he has obtained remarkable cures. He has often been consulted for blepharitis, for blepharokeratitis and particularly by adults for iritis and cyclitis. He observed a syndrome of the nasal nerve in two patients who both to his great surprise gave positive reactions to the Mantoux test. He recollects that nasal neuritis gives a syndrome characterized by (*a*) symptoms in the anterior pole of the eye (ulcerative keratitis, iritis), (*b*) symptoms of acute rhinitis and (*c*) ocular neuralgia, orbital, nasal or frontal. After giving details of the two cases mentioned, Charlin concludes that bacillary infection of nasal origin should be considered in all ocular disorders of obscure etiology and that this infection may exist without apparent signs, particularly in adults and particularly in females approaching the menopause.

S. H. MCKEE.

PRIMARY ACUTE OSTEOMYELITIS OF THE ORBIT. R. ONFRAY and F. MUNCH, Bull. Soc. d'opht. de Paris, April 1936, p. 311.

Primary acute osteomyelitis of the orbit is a rare condition which was described by Mackenzie and Lammelongue, and also by Rollet in the "Encyclopédie d'ophthalmologie." The case reported was particularly dramatic and severe and shows the importance of a precise diagnosis and early treatment.

A youth of 18 years was seized with sudden severe pain in the right orbit, extending around the orbital rim. This followed a light

cold. There was no especial discomfort in the nose. The next day the temperature rose to 40 C. (104 F.); the lids were red and swollen, and acetylsalicylic acid did not relieve the pain or the fever. After another twenty-four hours marked chemosis of the conjunctiva occurred; the eye was proptosed and fixed in position. The media of the eye were clear, but the disk was congested, and the veins were slightly tortuous. Examination of the sinuses gave negative results, as did that of the teeth. There was no evidence of thrombophlebitis. Pus suddenly pointed at the superior-internal angle of the orbit, where an incision was made. The subsequent course was stormy. Culture showed predominance of Staphylococcus. General complications were pleurisy and endocarditis. It was finally necessary to operate and secure complete drainage from under the periosteum.

L. L. MAYER.

TUBERCULOUS PANOPHTHALMITIS. A. B. KATZNELSON, Sovet. vestnik oftal. **9**: 205, 1936.

Tuberculous panophthalmitis is differentiated from other tuberculous infections of the eye by the predominance of exudative and purulent inflammation over the proliferation of specific tissue and by caseation and necrosis of tissue, with fistula formation. An active pulmonary process is present simultaneously.

This is the eighth case of this condition reported in the literature. [In a footnote Katznelson cites the case of a ninth patient, who died from meningitis after enucleation of the eye.—Abstractor.]

An 18 year old youth suffered from chronic pulmonary fibrous tuberculosis with an acute exacerbation—"pneumonia"—which was followed by loss of vision in the left eye during a period of two months.

Examination revealed moderate exophthalmos; in the lower temporal quadrant a yellowish round tumor was seen through the conjunctiva. The pupil was covered with a thin exudate; the iris was atrophic; tension was plus. A week later the granuloma softened, and on incision of the conjunctiva a cavity filled with necrotic tissue presented itself; it was connected with the vitreous. A guinea-pig given an injection of pus showed miliary tuberculosis at autopsy. A culture on egg medium yielded tubercle bacilli. A fistula formed at the site of the incision, and phthisis bulbi developed. The general condition and the process in the lungs improved after the fistula formed. The patient was discharged and began working.

Katznelson believes that all the toxic signs—the elevation of temperature, the loss of weight and the arthritis—were due to the "leading" active process in the eye in the form of subacute panophthalmitis with cavitation of the eyeball. The primary metastasis was in the choroid, where a solitary tubercle formed, which later showed conglomeration along the uveal tract. Detachment and necrosis of the retina followed; then necrosis of the vitreous and, finally, necrosis of the sclera with fistula formation occurred, so that true open tuberculosis of the eyeball was present in this case.

O. SITCHEVSKA.

Physiologic Optics

A New Theory of Anastigmatic Spectacle Lenses. F. Ostwalt, Arch. f. Ophth. **135:** 610 (July) 1936.

In several papers (*Bull. et mém. Soc. franç. d'opht.* **47:** 36, 1934; *Ztschr. f. ophth. Optik* **22:** 166, 1934; **23:** 132, 1935) Ostwalt modified his original theory of point-focal lenses. These modifications were criticized by Sundgvist (*Arch. f. Ophth.* **135:** 67, 1936). Ostwalt now replies and very convincingly states again the reasons why point-focal lenses should be constructed in such a way that pencils of rays which aim at the image of the center of rotation of the eye produced by the combination of the spectacle lens with the eye are free from the astigmatism of oblique incidence.

P. C. Kronfeld.

Physiology

On the Nature of the Ocular Fluids. K. Meyer and J. W. Palmer, Am. J. Ophth. **19:** 859 (Oct.) 1936.

Meyer and Palmer discuss the origin of the intra-ocular fluids, and report some experimental data. They give the following summary:

"Evidence for the dialysis theory of intraocular-fluid formation has been discussed. A polyuronic acid from the vitreous humor and its physico-chemical characteristics have been described. Highly viscous preparations of the acid have been obtained; its viscosity largely accounts for that of the vitreous humor. Evidence for the existence of this acid in the aqueous humor has been presented, which indicates that the intraocular fluid is at least partially formed by a specific secretion."

W. S. Reese.

Retina and Optic Nerve

Spasms of the Central Artery of the Retina. Jean Sédan and G. E. Jayle, Ann. d'ocul. **173:** 865 (Nov.) 1936.

The authors continue their article from the August 1936 issue of *Annales d'oculistique* and take up first the question of blindness. They discuss first binocular and then monocular blindness, illustrating the discussion with the histories of patients seen by them. From this they draw the following conclusions: Recurrent retinal spasm, whatever the cause, is always a transitory symptom of a local or general disturbance. It never has the tendency, even in the most intense forms, to provoke definite trouble.

The second part takes up mixed forms of visual troubles of spasmodic origin. These are subdivided into recurrent visual disturbances subsequently complicated by definite lesions, and prolonged and recurrent visual disturbances. Numerous examples are given in each subdivision. The article is to be continued.

S. H. McKee.

DETACHMENT OF THE RETINA: AN ARRANGEMENT FOR TRANSILLUMINATION OF THE SCLERA AND DIATHERMIA SIMULTANEOUSLY; AN EXPERIMENTAL CASE. J. LIJO PAVIA and M. DUSSELDORP, Bull. Soc. d'opht. de Paris, October 1935, p. 649.

The scleral lamp of Lange was equipped with diathermy electrodes and used in experiments on animals to show the method of localizing a hole and treating it at once with the current. Pictures of the instrument and of fundi of animals which had been operated on are included in the article.

L. L. MAYER.

METHODS IN THE ACTUAL TREATMENT OF DETACHMENT OF THE RETINA. P. BAILLIART, Bull. Soc. d'opht. de Paris, July 1935, p. 507.

Bailliart reviews the history of the treatment of detached retina. He believes that advance in the methods of treatment is shown by Weve's statistics on cases in which reattachments were obtained, viz.:

1930–1931.....	48%	1933.....	72%
1932.....	63%	1934.....	75%

He concludes that, as a rule, eyes with detachment of the retina should be operated on.

L. L. MAYER.

MEDICOLEGAL CONSIDERATION OF TRAUMATIC DETACHMENT OF THE RETINA. D. SABBADINI, Bull. Soc. d'opht. de Paris, July 1935, p. 520.

Professor Sabbadini, as the authority on legal medicine in Italy, presents the important facts on this subject as related to Italian law.

Gonin's review of the clinical varieties of traumatic detachment gives the principal theme for Sabbadini's discussion. The following have medicolegal value:

1. A written statement obtained at the earliest opportunity.
2. An early visit to a competent ophthalmologist.
3. A well identified type of traumatic reaction.
4. Evidence of trauma and its reaction.
5. Exclusion of slight trauma as a cause of detachment.
6. The possibility of fixing the time when a detachment is or is not definitely stabilized.

L. L. MAYER.

TRAUMATIC DETACHMENT OF THE RETINA. L. GENET, Bull. Soc. d'opht. de Paris, July 1935, p. 543.

Genet reports the case of a 16 year old patient to show the difficulty of deciding on the pathologic character of a traumatic detachment of the retina. At the time of injury the fundus could not be seen, owing to disturbance and clouding of the vitreous. Twelve days later the media were clear, but no retinal change was noted. Twenty days following the injury a detachment of the retina, with disinsertion superiorly, was noted.

Another patient, who was seen in consultation with Dr. Parifique, had a piece of metal strike the eye. He was taken to the hospital at once; because of the intense edema of the lids, little of the globe was seen. Four days later herniation of the iris and luxation of the lens (which was cataractous in part) into the anterior chamber were noted. Following iridectomy the lens absorbed, and when the fundus could be seen, four months following the injury, the entire lower portion of the retina was detached. Genet stresses the point that after injury of the globe hypotension or hypertension may ensue, followed or accompanied by detachment of the retina. In all operations for retinal detachment he has found that the subretinal fluid had been under tension. Iritic reaction following injury to the globe must make the prognosis more guarded; often a detachment of the retina is produced in these cases.

L. L. MAYER.

ETIOLOGY OF THE MACULA. H. HOEHNE, Ztschr. f. Augenh. **88:** 297 (March) 1936.

Hoehne describes the cases of three patients with macular coloboma who showed definite evidence that this was caused by an inflammatory process, probably tuberculous. He thinks that these cases should be separated from the group of cases of hereditary macular degeneration in which there is always a family history of similar disturbance and from cases of congenital faulty development of the macula in which there is usually evidence of other faulty development, especially of the eyes. The chief causes of the inflammatory type of macular coloboma he lists as acquired and congenital syphilis and tuberculosis.

H. GIFFORD JR.

CHORIO-RETINITIS SCLOPETARIA. T. H. LUO, Chinese M. J. **50:** 1405 (Oct.) 1936.

The changes in the eyeground which develop following gunshot injuries of the orbit are called chorioretinitis sclopetaria.

As the extensive free bleeding in the vitreous clears up, the following interesting and characteristic picture is revealed: The changes are essentially hemorrhages and whitish patches which involve wide areas in different parts of the fundus. The hemorrhages are preretinal, retinal, behind the retina or in the choroid and are mingled with the white patches in their neighborhood. The white patches are large and irregular but have circumscribed margins. They send out long processes in different directions in the fundus and are often interrupted by small round or oval sharply outlined light red areas.

The article has a colored drawing illustrating the condition.

S. H. MCKEE.

Trachoma

TRACHOMA. S. S. MEIGHAN and MARY URQUHART, Brit. J. Ophth. **20:** 201 (April) 1936.

This is an investigation into the question of the presence of Bacterium granulosis (Noguchi) in cases of trachoma in Glasgow. The

laboratory studies followed the general lines of those of Finnoff and Thygeson. Meighan believes that some of the discrepancies in the results obtained by various workers are due to the fact that some of these workers did not allow sufficient time for growth to progress to the extent of producing visible "colonies" before announcing that the micro-organism in question was not present in the material examined. Again, some of these investigators may not have been conversant with the fact that cocaine inhibits the growth of Bact. granulosis.

Meighan's studies showed that this organism was present in a proportion of the cases examined.

W. ZENTMAYER.

TREATMENT OF TRACHOMA IN CHILDREN. WIBAUT, Rev. internat. du trachome **13:** 174 (Oct.) 1936.

The prognosis for the cure of trachoma in children is much better than that for the cure of trachoma in adults. The requirements are early detection and prolonged and prudent treatment. Children under 5 years of age may show only suggestive thickening of the conjunctival epithelium, but between the ages of 5 and 10 years follicles are generally well developed. These follicles should be attacked by expression, and frottage with a 1 per cent solution of silver nitrate or some other suitable medicament should be continued for a few months thereafter. Strong caustics are to be avoided, and operation is seldom necessary.

J. E. LEBENSOHN.

THE LEUKOCYTIC FORMULA IN TRACHOMA. A. C. MIHAIL, Rev. internat. du trachome **13:** 193 (Oct.) 1936.

Examination of the blood of those affected with trachoma reveals a slight tendency to an increase of the mononuclear elements—lymphocytes and monocytes, especially the latter. Eosinophilia does not occur except with concomitant parasitic infection.

J. E. LEBENSOHN.

Tumors

EPITHELIAL CYST OF THE IRIS. J. CHARAMIS and SFALAGAKO, Arch. d'opht. **52:** 167 (March) 1935.

The case reported was that of an epithelial cyst with an actual cystic cavity the structure of which was the same throughout. There was no basal membrane, and the cyst was lined with polygonal and cylindric epithelial cells, each having a central nucleus. On the outside the epithelium was flattened and contained fusiform nuclei. Some iris tissue was adherent to it.

In the majority of cases epithelial cyst of the iris is traumatic in origin, developing at the site of a perforating wound of the cornea or, very often, after operation on the anterior segment of the eye. However, the traumatic cyst as a rule arises from corneal or conjunctival epithelium. There are other cases in which the cyst is nontraumatic and endothelial in origin. In still others it may be congenital, arising

from an ectodermic inclusion. Charamis and Sfalagako call attention to the value of biomicroscopic examination in cases of epithelial cyst of the iris but state that in the final analysis the diagnosis depends on microscopic examination after extirpation.

S. B. MARLOW.

BIZARRE GRANULOMA OF THE ORBIT. E. REDSLOB, Bull. Soc. d'opht. de Paris, April 1935, p. 257.

A 21 year old man had tearing and swelling of the lids of the left eye for three months. Vision was normal in both eyes. A slight chemosis of the bulbar conjunctiva and edema of the inferior lid of the left eye were noted. On palpation one could feel under the lower lid a firm mass, round but not painful. A horizontal incision along the border of the inferior lid revealed a round fatty mass. When the mass was elevated there appeared a well delimited reddish mass fused to the nasal bone. This was easily excised. There was no diplopia following the operation. Grossly the tumor formed a compact mass consisting of confluent dark and clear areas. Microscopically the hue of these areas seemed to depend on the color of the nuclei of the cells. The dark areas were formed by a mass of round cells having much chromatin in their nuclei. Lymphocytes and plasma cells were noted in the dark areas while epithelioid cells and indefinable material were found in the clear spaces. Because the dark areas surrounded the clear spaces the formation was that of a nodule. The muscle fibers were separated by the infiltration and in addition to being discolored were dissociated. Redslob believes the tumor to have been an inflammatory lesion and not a neoplasm. General examination disclosed nothing except an increase of the eosinophils of the blood to 7 per cent. Photomicrographs and a bibliography are included.

L. L. MAYER.

A CASE OF NEURINOMA RETINAE. C. F. M. PIECK, Arch. f. Ophth. **135:** 451 (July) 1936.

In an eye removed from a woman aged 75 with the diagnosis of retinal detachment and absolute glaucoma, Pieck discovered a primary malignant tumor of the retina which differed in many respects from those described in the literature. The tumor consisted of several types of cells, which seemed to be derived from ganglion cells, glia cells and nerve cells.

P. C. KRONFELD.

Uvea

ETIOLOGY OF HYALINE VERRUCOSITIES. J. BERNACKA-BIESIEKIERSKA and A. WIECZORCK, Bull. Soc. d'opht. de Paris, October 1935, p. 626.

The authors observed a family of seven children in which two girls and a boy had peripapillar hyaline verrucosities (drusen). Congenital syphilis was in the family, and only in those members having the alterations of the pigment of the retina and choroid characteristic of that disease were the hyaline deposits in evidence. Parsons and Morton considered such deposits as the result of a past inflammatory

process. Iwanoff felt that they were portions of the lamina vitrea of the choroid. Lauber explained them as portions or cells of the pigment epithelium of the retina detached during fetal life. Others maintained that the hyaline verrucosities were due to irregularities in the evolution of the ectoderm and mesoderm. Thus every factor having a bearing on the production of the eye has been given as causative. Three photographs are shown.

L. L. MAYER.

Vitreous

HYALOID CANAL WITH AREA MARTEGIANI AND SYMMETRICAL HYALOID FORAMEN. A. VOGT, Ztschr. f. Augenh. **88:** 1 (Dec.) 1935.

A case of retinal detachment is presented, with excellent illustrations. An oval opening was seen to be sharply delimited, lying almost over the optic disk. The edge of the opening lay from 6 to 8 diopters in front of the retina. Through the opening the retinal vessels were seen as clearly as through the retinal tear. Running out from the opening were sets of radial folds, which slightly obscured the retinal vessels. The folds were thought to be due to disturbance in the posterior hyaloid limiting membrane. The other eye, in which there was no retinal separation, showed a similar distinct oval opening in the vitreous. These openings were thought by Vogt to represent the posterior hyaloid foramen and prove the presence of the funnel-shaped area martegiani.

This case and experiments with berlin blue on the eyes of animals and young children by Gasser should prove conclusively the presence of the posterior hyaloid canal and foramen and would give an anatomic basis for ring-shaped separation of the posterior limiting membrane of the vitreous.

H. GIFFORD JR.

POSTERIOR RING-SHAPED SEPARATION OF THE VITREOUS. E. KRAUPA, Ztschr. f. Augenh. **88:** 224 (Feb.) 1936.

Kraupa describes and illustrates two cases of circumscribed holes in the posterior limiting layer of the vitreous as seen with the arc light ophthalmoscope. One was smaller than the optic disk and was placed eccentrically. The other patient showed a pretzel-shaped hole. Kraupa feels that, because of their shape and position, they have no relation to the area martegiani but represent destruction of the posterior limiting membrane of the vitreous, being merely one phase of senile ocular degeneration. He claims priority for describing this condition in 1914 and feels that these changes may either precede or be the result of retinal separation.

H. GIFFORD JR.

Therapeutics

DISEASES OF THE EYE TREATED WITH DIATHERMY. P. M. BATRACHENKO and T. M. ASKALANOVA, Sovet. vestnik oftal. **9:** 149, 1936.

Nine tables illustrate the results of the treatment in various diseases of the eye. In forty-five patients suffering from atrophy of the optic nerve of varied etiology, only those in whom some nerve fibers

were left intact were partial regeneration of these tissues and improvement of vision and of the visual fields obtained. In atrophy of long standing there was no response to the treatment. In fourteen cases of optic neuritis due to toxic doses of plasmochin and in eleven cases of malarial neuritis diathermy was definitely favorable, as the edema of the disk and retina disappeared and vision improved with this treatment. Diathermy is contraindicated in glaucoma because the intra-ocular tension is raised during the treatment; in four patients an attack of acute glaucoma was caused by the treatment. The albumin content of the aqueous of two patients with glaucoma was examined before and after the treatment; it was increased from 3.26 to 16.5 per cent.

Diathermy was of no value in choroiditis and retinitis pigmentosa. In eight cases of vitreous opacities the treatment gave definite improvement. Diathermy was definitely favorable in two of three cases of sympathetic ophthalmia: The intra-ocular tension was raised, and synechiae were torn loose.

Diathermy is contraindicated in trachoma, as it causes an increase of secretion, hyperemia and the appearance of new blood vessels.

In fifty-nine patients suffering from malarial, arthritic and gonorrheal iritis and iridocyclitis, diathermy acted almost as a specific treatment, as the pain subsided after four or five treatments and formation of synechiae was prevented. Tuberculous and syphilitic iritis did not respond well to the treatment. In eleven cases of traumatic iritis and iridocyclitis, diathermy caused exacerbation of the inflammatory process, with increase of the number of precipitates.

Eighteen patients with incipient cataract responded favorably to diathermy treatment: the vision was improved and remained so during one year. Further study and observation are being conducted.

In scleritis and episcleritis diathermy was definitely indicated, as the duration of the disease was shortened and the bluish nodules disappeared with this treatment. This method of treatment was also successful in cases in which grafts after Thiersh were made and in cases of plastic operations on the lids.

In keratitis (forty-seven cases) of malarial origin, parenchymatous, neuroparalytic and tuberculous, the results were not favorable.

O. SITCHEVSKA.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Clinical Meeting, Dec. 11, 1936

MR. W. H. MCMULLEN, O.B.E., F.R.C.S., *President*

VASCULAR LESIONS FOUND IN ASSOCIATION WITH MYOPIA: A REPORT OF SIX CASES. MISS J. M. DOLLAR.

CASE 1.—A woman aged 60 was first seen six years ago because of extensive nebulae in the right cornea and myopic changes in the left fundus. A recent examination disclosed visual acuity of 6/24 in the left eye with a minus cylinder, axis 180. In the region of the left macula there was a black circular area, which seemed to be an example of Fuchs' black spot, in spite of the low myopia. The coagulation time was three minutes.

CASE 2.—Retinal hemorrhage associated with high myopia was present in a woman aged 38. When she was first examined, in November 1917, corneal nebulae were seen, due to old interstitial keratitis. In January 1926, there was a hemorrhage in the macular region of the right eye. Eight years later vision with glasses was 6/12 in the right eye and 6/18 in the left. Two years later, in September 1936, she complained that vision of the left eye had deteriorated; with the aid of her glasses she could count fingers at the distance of 1 foot (30.5 cm.). The physician reported that the condition was mild pituitary dystrophy. The Wassermann reaction was negative; the coagulation time was two and one-half minutes. There was a whitish excavated area in the right fundus, at the site of the first hemorrhage.

CASE 3.—A woman aged 44 complained of failing vision in the left eye. In each fundus there were myopic changes. Vision of the right eye was 6/12 and that of the left eye was 6/60 with a minus 9 sphere for each. There was a coagulation time of eight and one-half minutes. The point of interest in this case was an absorbing choroidal hemorrhage in the left eye downward and outward from the disk, in association with high myopia.

CASE 4.—A woman aged 60 complained of poor vision. Choroidal hemorrhages were seen downward and outward from the disk in the left eye, associated with a high degree of myopia. In each fundus myopic changes could be seen. The blood pressure was 120 systolic and 80 diastolic.

CASE 5.—A man aged 49, who was first seen four months ago, complained that vision of the left eye was getting worse. Vision of the right eye with glasses was 6/6; that of the left eye was 6/36. There were retinal hemorrhages and exudates in both fundi. The blood urea

content was 48 mg. per hundred cubic centimeters, but there were no renal casts. The systolic blood pressure was 260; the Wassermann reaction was negative. In this case there were advanced renal changes combined with an absence of definite evidence of renal involvement, as is usual in cases of essential hypertension.

CASE 6.—The case of a woman aged 66 with retinitis circinata was reported. When seen a month ago she said that the sight of the left eye had been getting worse for years. With glasses she could only count fingers with the left eye. No thickening of the radial arteries was evident; the blood pressure was 120 systolic and 60 diastolic. This patient showed the typical lesion.

MACULAR CHOROIDAL HEMORRHAGE: MR. R. M. TRYELL.

CASE 1.—A married woman aged 46 had loss of central vision in the left eye for six weeks. A large mound of one and one-half disk diameters was seen lying above the macula, with a retinal vessel crossing it. The center of the mound has since caved in, leaving an area with a crater-like appearance. Vision improved from 6/36 (at the first attendance) to 6/12.

CASE 2.—Probable macular choroiditis was shown by a woman aged 37. She complained of a central patch of blindness in the left eye, but vision around it was possible. Examination showed the exudative type of choroiditis at the macula but little disturbance elsewhere. In front of the macula was a good deal of exudate. The Wassermann reaction was negative. Several diseased teeth were extracted.

CASES EXEMPLIFYING VARIOUS TYPES OF RETINITIS PIGMENTOSA. MR. JOSEPH MINTON.

CASE 1.—A woman aged 31 complained of night blindness. Vision of each eye was 6/12, but the field of each eye was concentrically constricted to 10 degrees. The disks were yellowish; the retinal arteries were narrowed, and in the periphery a few specks of pigment were visible. The patient's general health was good; there was no history of consanguinity in the parents, and no other members of the family showed ocular abnormalities.

CASE 2.—A woman aged 28 complained of night blindness, which she had noted for three years. Both eyes showed full vision, but the right and the left field were concentrically constricted to 15 degrees. The retinal arteries were narrowed, and one could see the choroidal circulation. Deposits of pigment were scattered in the periphery. The quantity of pigment had been increasing. The general health was good, and there was no history of consanguinity.

CASE 3.—A girl aged 8 years complained of night blindness. Vision in both the right and the left eye was 6/9; the right and the left field were concentrically constricted to 10 degrees. The retinal arteries were much narrowed, and the choroidal circulation was visible. In each fundus fine granules of pigment and large pigmented areas were seen. In February 1935 the patient showed white shining bodies in

the periphery of the fundi and a much smaller amount of pigment. The Wassermann reaction was negative, and the general health was good. No abnormalities were found in the remainder of the family.

OXYCEPHALY IN A PATIENT SHOWING APERT'S SYNDROME. MR. S. R. GERSTMAN.

A boy aged 7 years had a broad, square skull, bilateral ptosis, ankyloblepharon and syndactylia, the second and third, and the fourth and fifth toes being webbed. There was no consanguinity of the parents, but the familial relationship of the syndrome was shown by the syndactylia of the second and third toes of both feet of a female cousin of the patient's father. Vision was good; there was no atrophy of the optic nerve, and the boy was unusually bright.

OXYCEPHALY IN A PATIENT SHOWING CROUZON'S SYNDROME. MR. S. R. GERSTMAN.

A boy aged 12 had oxycephaly and prognathism, a high-arched palate, exophthalmos, coloboma of the irides and atrophy of the optic nerves. Vision without glasses was 6/60 in each eye; vision with glasses was 6/36 in the right eye and 6/18 in the left. The patient was somewhat dull for his age. The maxillae were ill developed.

FAMILIAL PERIPAPILLARY CHOROIDAL SCLEROSIS IN A PATIENT WITH AN EXTENSIVE CONDITION INVOLVING THE CENTRAL AREAS (LAURENCE-MOON-BIEDL SYNDROME). MR. H. AVERY and MR. ARNOLD SORSBY.

A boy aged 16, the fifth son of parents who were first cousins, showed the complete Laurence-Moon-Biedl syndrome, i. e., obesity, hypogenitalism, polydactylism, pigmentary degeneration of the fundi, mental deficiency and a recessive character of the disorder. There were five children of the marriage, all sons. The second son, who had been affected in the same way, died of pneumonia last year at the age of 23.

CORNEAL DYSTROPHY. MR. E. J. SOMERSET.

A boy aged 8 years, since the age of 16 months has had twenty-three attacks of spontaneous erosion of the cornea, characterized by superficial loss of corneal epithelium over an area 6 or 7 mm. in diameter. At the time of erosion there was little injection, and in a few weeks the area had become healed over. Examination showed a very few dotlike opacities in Bowman's membrane.

STARGARDT'S DISEASE. MR. L. H. SAVIN.

A girl aged 15 showed typical pigmentary macular degeneration, without other abnormal signs. The other members of the family were healthy, and there was no consanguinity. In 1932 vision was 6/12; central vision is now lost.

FAMILIAL DISTICHIASIS. MR. L. H. SAVIN.

The cases of two patients with familial distichiasis, aged 3 and 2 years, respectively, were reported. The first patient was greatly troubled by pain and photophobia; therefore the posterior rows of lashes were excised and the raw areas on each lid were filled by grafts of mucous membrane from the lip. The photophobia is now relieved. Suggestions were sought for treating the patient's sister, whose lashes are not yet causing trouble.

DISCUSSION

MR. CHARLES GOULDEN: I should advocate electrolysis, half a dozen lashes being treated at a time.

RUPTURE OF DESCEMET'S MEMBRANE OF THE LEFT CORNEA SUSTAINED AT BIRTH. MR. L. H. SAVIN.

A boy aged 2½ years, who was delivered by instruments, showed a scar much less marked than formerly, especially in the posterior layers of the substantia propria of the left cornea. A less obvious linear indentation of the left frontal bone was also present.

REVOLVER SHOT THROUGH BOTH ORBITS. MR. L. H. SAVIN.

A married woman aged 28 accidentally discharged a revolver into the right temporal region, and the bullet emerged through the left eyebrow. When first seen the patient was conscious but could not see. Both eyes were proptosed, and both vitreous chambers were full of hemorrhage. Multiple fragments of bullet were revealed in the ethmoid bone by the roentgen rays. On recent examination the vitreous hemorrhages had cleared, leaving the right eye blind and with massive retinitis proliferans. The left fundus showed gross retinitis proliferans, but a little vision had been recovered in that eye. A callus from the injured right zygoma involved the right temporal muscle, limiting the opening of the mouth.

DISCUSSION

MR. CHARLES GOULDEN: I think the prognosis as regards sight is hopeless. I saw a number of somewhat similar cases in the World War, and the outlook was grave.

OPHTHALMOPLEGIC MIGRAINE. MR. S. R. GERSTMAN.

A woman aged 48 has been having recurring attacks of migraine ever since her fourteenth year. At the time of the first attack the right eye diverged, and it has not since returned to the normal position. In 1935 she had a severe attack and was hospitalized; she was found to have complete palsy of the third nerve on the right. Recent examination showed some weakness of muscles served by the third nerve. The ptosis had completely disappeared. With correction, vision was 6/12 in the right eye and 6/6 in the left.

NEOPLASTIC TYPE OF DISCIFORM DEGENERATION OF THE MACULA. MR. ARNOLD SORSBY.

A woman aged 74 in 1931 had in the right eye with a plus 1 sphere vision of 6/6 and in the left eye vision of 1/60. The left central

area showed disciform degeneration of the "neoplastic" type, with hemorrhages. In October 1933 the left eye showed choroidal degeneration. In July 1935 vision of the right eye was less than 6/60. There were a hemorrhage into the vitreous and, in the left central area, an atrophic scar. In September 1936 the hemorrhages had disappeared from the right eye, and the swelling was more marked.

MACULAR DEGENERATION IN A PATIENT AGED TWENTY-FOUR. MR. FREDERICK T. RIDLEY.

A case of pure macular degeneration, beginning in the early twenties, was presented. Vision was not yet involved, although definite ophthalmic changes were noted. The mother, aged 52, with vision of 6/18, showed a similar condition. The condition was, therefore, hereditary, and was probably a dominant lesion.

Jan. 8, 1937

MR. W. H. MCMULLEN, O.B.E., F.R.C.S., *President*

MELANOMA OF THE IRIS. MR. FRANK A. JULER.

A man aged 70 came for glasses. He had not noticed the discoloration of his iris. Examination revealed a tumor affecting two thirds of the width of the stroma of the iris and protruding forward into the anterior chamber, especially below the pupillary margin. There was a slight pulling of the pupil downward with slight ectropion uveae at the pupillary margin.

DISCUSSION

MR. CHARLES GOULDEN: A point of importance in diagnosing malignant disease is the deformity of the iris. The shape of the iris is not deformed by a nevus, but it is deformed by a malignant growth. In this case of Mr. Juler's, as in the last case of [illegible] condition that I observed, the root of the iris is free from growth. I successfully removed the tumor in my case by iridectomy, and there had been no recurrence several years after the removal.

MR. LEIGHTON DAVIES, Cardiff: I have observed two or three cases somewhat similar to this, but the patients were persons with dark irides. I have watched them for several years, and in each case there was an elevation of the tumor above the surface of the iris, without change in appearance for three or four years. My view about the condition in the patient now shown is that the growth is not malignant. If it is malignant, it is probably a melanotic sarcoma. If this is a case of melanotic sarcoma, I do not think that one can hope to save the patient's life by removing a portion of the iris.

MR. A. F. MACCALLAN: In the last few years I have shown, at meetings of the section, two patients with a condition similar to that of Mr. Juler's patient, and in them the condition has remained the same for many years. In my opinion, the growth in Mr. Juler's patient is a mole, not a malignant tumor.

MR. W. H. MCMULLEN: The fact that the patient's wife had not noticed the defect of the iris suggests that it is not new. I think that the arrangement of the uveal pigment in this case is against a malignant growth. My advice would be to wait and from time to time measure the size of the pigmented mass.

HEMISPHERICAL FORMATIONS IN BOWMAN'S MEMBRANE. MR. EUGENE WOLFF and MR. T. KEITH LYLE.

A case of idiopathic epilepsy is presented in a stevedore aged 32. When 17 years old he began to suffer from dizzy spells, and at 21 years he had attacks of petit mal, followed at a later date by attacks of grand mal. Sometimes he has as many as three or four attacks a day. Examination showed that vision in the right eye was 6/6 and the fundus was normal. When 15 years of age he sustained an injury to his left eye with a tennis ball, and since then vision in the left eye has been blurred. At 23 years of age the sight of the left eye became much worse, a black film appearing to spread over that eye from the inner margin, and within three years he had become blind in that eye. The tension of the left eye was plus 2. The cornea was edematous, and the lens was slightly opaque. The optic disk was cupped; the media were hazy, and there was an extensive detachment of the retina. Treatment, which was carried out last year, was excision of the left eye, with the insertion into Tenon's capsule of a fat graft taken from the anterior part of the abdominal wall.

In regard to the pathologic aspect, abnormal, hemispherical and homogeneous structures were found in Bowman's membrane, occupying that portion which is next to the epithelium. These structures were actually in, not on, Bowman's membrane, and that membrane was not pushed backward, as was evidenced by its straight posterior border. The structures varied in size up to about 10 micromillimeters at their bases. Some sixty could be seen in each section, and there were more toward the central than toward the peripheral parts of the cornea. The hemispherical formations stained red with hematoxylin and eosin and orange-red with the Van Gieson stain. That the structures did not consist of amyloid was shown by the fact that they did not give the characteristic stain with methyl violet or congo red. It is probable that they were hyaline, because they stained deep blue with Kühne's stain. Probably they were produced by degenerative products of the corneal epithelial cells or by degeneration of the perforating nerves.

EXPERIMENTAL STAINING OF THE RETINA DURING LIFE. MR. ARNOLD SORSBY, DR. A. ELIOT. DR. G. W. GOODHART and DR. I. B. MORRIS.

Our problem in this investigation was to determine whether we could make the translucent normal retina visible ophthalmoscopically by vital staining during life. Vital staining has been applied to other tissues, both for histologic purposes and as a therapeutic measure. An example of the latter is the use of various vital dyes in leprosy. The difficulties in vital staining of the retina are bound up with those which

have been encountered in staining the nervous system as a whole. There appears to be a barrier between blood and nerve tissue, a holding back of many substances circulating in the blood, preventing them from reaching the cerebrospinal fluid and the brain. The mechanism of this barrier has been the subject of much dispute. The blood-brain barrier prevents entry into the brain not only of various colloids which are found in the blood stream but also of toxins and such administered medicaments as arsenic. In this way the blood-brain barrier acts as a mechanism for the protection of the brain. This mechanism, however, is a detriment in some conditions, such as cerebrospinal syphilis, as it effectively prevents the therapeutic agent from coming into contact with the brain.

The existence of this blood-brain barrier has been studied through observing the action of various dyes which had been injected intravenously. Since the classic experiments carried out by Goldmann in 1913, it has been the prevailing view that the choroid plexus plays a dominant part in this mechanism, because trypan blue, the dye used in these experiments, was found mobilized in the plexus, while none of it was found in the brain tissue itself. The existence of the blood-brain barrier has been regarded as an insurmountable difficulty in vital staining of the brain. The same mechanism which prevents vital staining of the brain was presumably effective in preventing staining of the retina. Such work as has been done on vital staining of the eye has dealt largely with demonstrating the mechanism of the formation of the aqueous by the ciliary body. Recent work has shown that the blood-brain barrier is effective for acid dyes only, and basic dyes have been proved to penetrate the barrier and to stain the brain. Experiments carried out with acid dyes failed to produce any staining of the retina. The injection of basic dyes gave results which were difficult to interpret, since all the basic dyes are highly toxic and any staining that was obtained might well have been a supravital phenomenon, and held out little promise for clinical application.

Further work consisted of a search for a nontoxic dye which would pass the blood-brain barrier and produce the desired staining of the retina. Light green S. F. was found to answer the purpose, as it was nontoxic to the rabbit and produced transient staining of the fundus when injected intravenously. Staining of any duration clearly affecting the retina could not be obtained in the healthy rabbit. Degeneration of the rabbit's retina was then experimentally produced by the intravenous injection of sentojod, which is a solution containing sodium iodide, sodium iodate and iodine; when such rabbits were subjected to the injection of light green, the same transient staining as that noted in normal rabbits was obtained at the time of the injection. Half an hour later the fundus of these rabbits showed an intensive green coloration, which was of irregular distribution. This could not be obtained in the normal rabbit, and this failure was, presumably, because the normal rabbit's retina converts the dye into a leukobase by oxidation, the damaged retina doing so to a much more limited extent. Further investigations have shown that the same results can be obtained by intramuscular injection of the dye. The histologic investigations showed that the dye was situated in the retina.

DISCUSSION

MR. EUGENE WOLFF: Some time ago there appeared in the ARCHIVES an article reporting a series of cases of some cutaneous disease in which the fundus was stained yellow, and in those cases, I think, it was decided that the dye was in the vitreous. The impression given in those cases was that the observer was seeing the retina through the dye in the vitreous. It is difficult to exclude that possibility, and I ask whether any of these animals referred to by Mr. Sorsby were looked at with the eye opened and the vitreous removed.

MR. ARNOLD SORSBY: Mr. Wolff is referring to a case in particular which was published in the September 1936 issue of the ARCHIVES (Gerber, A., and Lambert, R. K.: Blue Appearance of the Fundus Caused by Prolonged Ingestion of Methylthionine Chloride, ARCH. OPHTH. **16:** 443 [Sept.] 1936), accompanied by a colored plate showing a blue fundus. Four patients with urogenital tuberculosis were given methylthionine chloride (methylene blue) for four years; the fundus assumed a blue color. That the coloration was due to staining of the vitreous was a conjecture and not a fact. My collaborators and I have opened a number of eyes; we found that the vitreous was always unstained, the lens was always unstained and the aqueous was sometimes stained and sometimes not stained. The problem is to find a group of dyes which get past the choroid plexus and reach the brain, i. e., its nervous tissue, which dyes shall be nontoxic and shall not cause local damage.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Dec. 21, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

A CASE OF OXYCEPHALY. DR. GIROLAMO BONACCOLTO.

A boy aged 13, with a negative family history, born at the end of a full term pregnancy, presented an abnormal skull which was classified as oxycephalous. The following signs were found: a characteristic roentgen appearance of convolution depressions on the walls of the skull and shortening of the anteroposterior diameter of the orbit; exophthalmos of 27 mm.; divergent strabismus; primary atrophy of the optic nerve with loss of vision in both eyes and a corresponding contraction in the visual fields. Accompanying the report was a review of the literature.

MALIGNANT MELANOTIC TUMOR OF THE LIMBUS. DR. ROWLAND H. MERRILL.

A case was demonstrated of malignant pigmented tumor of the limbus occurring in a white woman of 30. A large pigmented non-

movable nodule was present at the temporal limbus of the right eye. A highly vascular, succulent, infiltrative mass extended from the nodule around the limbus for half its circumference and invaded the superficial layers of the cornea in a band about 3 mm. wide. A small chain of metastatic nodules occurred in the conjunctiva 5 mm. below the cornea. Vision in each eye was 20/70, and was corrected to 20/30+. The tension was normal. Both lenses showed coronary cataracts. The fundi were normal.

DISCUSSION

DR. ALGERNON B. REESE: I doubt whether enucleation will prove sufficient in this case, because it seems to me there is extensive involvement of the conjunctiva. If all the involved conjunctiva were removed at the time of the enucleation, I do not believe there would be a sufficient amount left to cover the socket. In the cases I have observed, as a rule, there is rather extensive involvement of the basal layer of the conjunctiva, even farther away than the obviously involved areas. Therefore I believe that exenteration is the procedure indicated.

KERATOPLASTY AND IMPROVED INSTRUMENTS. DR. R. TOWNLEY PATON.

Rabbits' eyes afford the most economical and best material for experimentation. Metal stencils or a double-bladed knife with angulated blades may be used to outline grafts in certain cases. For eyes with a shallow anterior chamber and iris bombé, a broad double-edged knife, concave on one surface, may be found advantageous.

DISCUSSION

DR. RAMON CASTROVIEJO: Most of the ophthalmologists who have had any experience in making corneal transplants have been more interested in reporting success than in finding out causes for failure and ways to prevent this failure. It is difficult to state at the present time which of the methods of corneal transplantation now used is the best. It may happen, as in any other field of surgery, that the operator accustomed to use a particular method will obtain with this method better results than if he were to use other methods with which he has had less experience. Therefore, I am sure that Elschnig, using the old von Hippel technic of corneal transplantation, will have better results than if he were to use Thomas', Filatov's or my method, and vice versa.

At the Institute of Ophthalmology of the Columbia-Presbyterian Medical Center seventy corneal transplantations have been performed. After this series of operations it was possible to tell beforehand those cases in which the condition was favorable for corneal transplantation and those in which it was not. The results obtained in the last five months have corresponded with the preoperative diagnosis. As a rule, in the cases in which the condition was diagnosed as unfavorable the transplant became either nebulous or opaque. Of the cases in which the condition was diagnosed as favorable, in more than 90 per cent the transplant remained permanently transparent.

Dr. Paton has mentioned the difficulty in diagnosing anterior and posterior synechiae when the cornea is densely opacified. This problem has been taken up by some authors. Thomas and I have described

methods of transillumination which render easy the diagnosis of anterior and posterior synechiae in cases of very dense leukoma.

Dr. Paton has also said that in some instances, especially when the knife is dull, he uses the device that he has shown tonight. This device when applied to the cornea makes it easy to outline in this structure either a square or a triangle. It should be remembered that before operation for corneal transplantation the knife should be tested to make sure that it is in good working condition. No one would think of starting an operation for cataract before making sure that the knife used to make the incision was sharp. The incisions made to outline the flap to be transplanted should be clean. Therefore the knife must be sharp; otherwise, irregular edges of the transplant and cornea of the host will be obtained which may defeat the success of the operation.

To outline a flap my associates and I use a knife with blades made from razor blade material. It is very sharp and is generally used in no more than two or three operations. The knife is tested before operation in the same manner as the knife used to make incisions for operations for cataract.

Dr. Paton has shown some diagrams illustrating a knife about 4 mm. in width, which goes through the anterior chamber. The puncture seems to be located in the cornea about 2 mm. from the limbus, and the counter-puncture is at the same distance from the limbus on the opposite side. With this knife, Dr. Paton stated, injury of the iris and lens may be prevented during dissection of the corneal leukoma. The idea expressed by Dr. Paton has been worked out practically by Dr. Filatov, who, after making the puncture and counter-puncture as described by Dr. Paton, introduces into the anterior chamber an ivory plate that he calls a prophylactic spatula. Dr. Filatov has been using this prophylactic spatula for some years and reported a number of successful results with it, but recently I have been told that he is no longer using the prophylactic spatula because the combination of knife and spatula caused, in some instances, injury of the iris and lens, which is the very thing that he was trying to prevent.

Dr. Paton has presented a double-bladed knife with the blades placed at a certain angle. He claims that shelving may be obtained with this while one is outlining the corneal flaps. It is difficult to understand how with a double-bladed knife in which the distance between the blades does not vary while the incision is made it is possible to obtain a shelving. The shelving claimed to be obtained by Dr. Paton is a mechanical impossibility.

For the past two years my associates and I have been experimenting with a new type of trephine made of a suction spoon which fastens to the cornea and a rotating blade placed at an angle of from 5 to 45 degrees which, at the same time that it rotates around the suction spoon, penetrates deeper and deeper. So far two such trephines have been completed. The last one worked satisfactorily in normal corneas but did not perform well when the resistance of the cornea had been increased by proliferation of connective tissue. More work is being done to overcome the difficulties encountered, and I am presenting the idea in the hope that some of the members might aid in finding the solution. It is gratifying to know that more persons every day are interested in keratoplasty, a problem which, when solved, will benefit so many unfortunate ones.

DR. R. TOWNLEY PATON: Dr. Castroviejo has given this subject a great deal of thought, but I believe that he forgets the important fact that there is considerable upward pressure of the cornea against the twin-bladed knife, and a slight bulge is produced, part of which is compensated for by the angulation of the blades. The operator should on no account attempt to penetrate through the entire thickness of the cornea with this knife. Another advantage of the double-bladed knife which I forgot to mention is that a more clearly outlined window may be obtained with this instrument, owing to the slight tearing action of the angulated blades as they are passed over the epithelial surface of the cornea.

THE DIAGNOSTIC SIGNIFICANCE OF EPINEPHRINE WHEN APPLIED BY INSTILLATION INTO THE CONJUNCTIVAL SACS IN THE STRENGTH OF 1:1,000. DR. LOUIS HUBERT.

This article will be published in full in a later issue of the ARCHIVES.

CORRECTION OF COMBINED VERTICAL AND LATERAL STRABISMUS. DR. CHARLES E. DAVIES.

A case was presented in which there was complete correction of esotropia of 75 centrads associated with hyperphoria of 3 centrads in the primary position and hypertropia of 26 centrads on looking up and to the left by a single recession of the right internal rectus muscle and resection tenotomy of the right inferior oblique muscle.

DISCUSSION

DR. JAMES WATSON WHITE: Dr. Davies has shown an excellent result of an operation for combined vertical and lateral strabismus.

There is one phase of this question which Dr. Davies had no cause to mention but which I have failed to emphasize enough. When the eye that has the paretic superior rectus muscle is used to fixate, the inferior oblique muscle has a secondary deviation which usually results in hypertrophy. In this case tenotomy of the inferior oblique muscle was indicated. However, when the nonparetic eye is used to fixate, no hypertrophy develops, as constant overstimulation is not present. When this condition is present, resection of the paretic superior rectus muscle is indicated.

OPERATION FOR RETINAL DETACHMENT. DR. JOSEPH LEVINE.

It is suggested that in localizing the retinal tear the ora serrata be used as the fixed point and not the optic disk, because the distance from the limbus to the optic disk depends on whether the temporal or the nasal side of the globe is measured. A curved millimeter rule is recommended, as advocated by Vogt, in measuring distances on the globe. To set up adhesive choroiditis an electrical current (a combination of cutting current and coagulation current) of a wavelength of 300 meters is used. Between 30 and 40 milliamperes are applied through individual steel pins which are inserted into the globe about 4 mm. apart. A barrage is made around the tear and also over the region of the detachment. A small opening is made with the trephine over the most

pendulous portion of the detachment, and the choroid is punctured to allow drainage of the subretinal fluid. The individual pins are then removed.

SUMMARY OF ORTHOPTIC WORK AT THE MANHATTAN EYE, EAR AND THROAT HOSPITAL. DR. FRANK CONRAD KEIL.

Approximately 500 patients with strabismus were examined in the orthoptic clinic of the Manhattan Eye, Ear and Throat Hospital during the past two years. This summary is in no respect a record of research work. Each case was assigned to one of the following groups:

Group	Type of Strabismus	Number of Cases
A	Monolateral convergent concomitant	377
B	Alternating convergent concomitant	70
C	Divergent concomitant	85

GROUP A.—After wearing glasses from two to three weeks, 99 patients had fusion at orthophoria, and 17 had fusion and normal adduction but limited abduction. Fourteen with gradual reduction showed second degree fusion after from three to four months. With glasses, all had at least second degree fusion with occasional stereopsis, normal duction and no suppression, amblyopia or abnormal retinal correspondence. Without glasses, esophoria of from 10 to 40 degrees was present. Of 51 patients who had orthoptic training (given for one-half hour periods twice a week over a period of from six to twelve months), 11 could fuse at orthophoria; 4 fused with prisms base out of 10 degrees, and 1 had unstable fusion. Three had improved fusion and amplitude, but high esophoria remained with glasses. There were 14 who had doubtful fusion and required prisms base out for binocular vision. Thirty-three made too few visits or were uncooperative.

GROUP B.—Ten patients with deviation of less than 10 degrees had second degree fusion with glasses. Eleven with deviation of 40 degrees or more received preoperative training. Of the 20 who had postoperative orthoptic training, 9 had unstable second degree fusion, although 7 of these had preoperative training. Five whose vision was slightly overcorrected fused after training, and the remainder, some of whom had overcorrected vision, and others, undercorrected vision, had no binocular vision. Twenty-four failed to cooperate, and 2 had alternation of fixation.

GROUP C.—Seventy of the 85 patients were hyperopic. Only those with marked exotropia applied for treatment. The slight exophoria characteristic of myopia was probably improved with glasses. Seventeen persons with divergence excess of from 6 to 30 degrees had improved tolerance for fusion after orthoptic training. Eighteen who had vision characterized by high deviation, associated hyperphoria, little or no binocular perception, remote near point of convergence, which were frequently periodic, and cosmetic defects, were not aided by orthoptics. Five who had postoperative training had good fusion. The remaining patients who had postoperative training and who had under correction or overcorrection showed no improvement.

An orthoptic department may be considered as a "clearing house" for cases of strabismus. It has a major place in the education of parents in respect to the treatment of the "cross-eyed child." Its follow-up has been valuable in maintaining the treatment of amblyopia. The results of preoperative and postoperative orthoptic treatment have been encouraging, and the painstaking procedure used in examination has been helpful in guiding the surgeon to a more perfect solution of a difficult problem.

DISCUSSION

DR. LEWIS WEBB CRIGLER: The essentials of the treatment of strabismus are as follows:

1. A child with strabismus must be brought to the ophthalmologist as soon as the squint is detected.

2. The refractive error must be carefully and fully corrected.

3. Treatment must be begun at once by excluding vision from the fixing eye.

Nothing will be accomplished by muscular exercises or fusion training if vision in the squinting eye is not materially improved.

It is a waste of time to attempt to correct squint by orthoptic methods in cases in which there is a complete absence of the fusion sense, and in the vast majority of cases squint in time will be relieved merely by the use of proper glasses, provided the fusion sense is present. The sphere of usefulness of the synoptophore and similar instruments is in those cases in which the fusion sense has been suppressed but is not entirely lacking. I question the efficacy of such treatment unless it is carried on more intensively than it is now practiced, but the stumbling block immediately arises in the failure to obtain proper cooperation.

The whole subject is a perplexing one the answer to which has not yet been completely solved. Good has been accomplished in that ophthalmologists have been aroused to a greater sense of obligation to the squinting child. The chief advance that has been made is that one is better able to separate the surgical from the nonsurgical cases.

DR. JOSEPH I. PASCAL: I think it is good practice to start fusion training at the earliest possible time, even when vision in the squinting eye is only 20/200. Vision of 20/200 means the ability to recognize an object which subtends an angle of about 50 minutes. Most objects in the child's environment—his playthings and eating utensils, for example—and, for that matter, things in the adult's surroundings, subtend an angle of 50 minutes or more. These objects are recognizable by the amblyopic eye, and there is no reason why the eyes should not be trained to fuse the recognizable images, if there is any fusion faculty capable of being developed. As a matter of fact, trying to develop fusion from the beginning, say, as soon as vision is 20/200 in the squinting eye, helps to develop the vision of that eye. The very procedure used in trying to build up the working together of both eyes has a beneficial effect on the amblyopic eye.

There is one other point I wish to touch on. Dr. Keil in his report mentioned few cases of so-called false projection and abnormal retinal correspondence. These are phases of orthoptic training that should be thoroughly investigated. Abnormal retinal correspondence, as the term is now generally used, means primarily the habit of fusing the macular

image of the fixing eye with the nonmacular image of the deviating eye. Dr. Pugh in her recent book "Squint Training" stated that about 50 per cent of squinting patients have abnormal retinal correspondence. Worth, after a study of 2,000 cases, said that such a condition is rare. He called the nonmacular portion of the retina, the image of which is projected so that it would be fused with the macular image of the fixing eye, a "false macula." This term is now used differently. But the important fact is that he found this condition to be rare, though he found what he termed false projection to be common. Furthermore, this condition, which is really abnormal binocular vision (the fusion of a macular image with a nonmacular image) is scarcely mentioned by the French writers, notably by Drs. Cantonnet and Filliozat in their latest book "Strabismus," and they also have studied an enormous number of cases. The presence of such an extraordinary variance among observers suggests the need for a careful checking up on the condition.

Miss Dorothy Shadd: As to requiring visual acuity of 20/70 for the beginning of training, I agree that it is possible with large objects to get fusion with much lower visual acuity than that with which one can get fusion when small objects are used; one can get fair fusion with vision of 20/20 in one eye and of 20/200 in the other, but there is apt to be frequent suppression of vision in the amblyopic eye. That is the reason I have taken visual acuity of 20/70 as the requirement for duction training.

My associates and I observe a large number of persons with abnormal retinal correspondence among those seen in the orthoptic clinic, but their records do not appear in our slides because these patients are seldom in the groups who show normal fusion or duction. They do not easily acquire normal fusion or duction. In general, I find that abnormal retinal correspondence occurs in the group with monolateral convergent strabismus in conjunction with definite amblyopia.

Dr. John M. Wheeler: May I ask what is meant by duction?

Dr. Frank Conrad Keil: Duction is a term used in orthoptic training to indicate the action of fusion. Its extent is measured by the amplitude of fusion with prisms base in or base out.

Lesions of the Fundus in Polycythemia. Dr. Martin Cohen.

This article will be published in full in a later issue of the Archives.

Book Reviews

Wills Hospital Eye Manual for Nurses. By Gladys E. Cole, R. N., Chief Nurse, Wills Eye Hospital. Price, $1.75. Pp. 202, with 97 illustrations. Philadelphia, W. B. Saunders Company, 1936.

Miss Cole aims in this small manual to acquaint nurses with what they should know about the eye in practicing ophthalmic nursing. The book contains chapters on the anatomy of the eye and a short description of the diseases of the eye which have been observed by ophthalmologists of the Wills Hospital.

The important parts which deal directly with the nurses' activities are grouped under the following headings: irrigation of the eyes and the instillation of drops; examination and treatment of patients with ocular disorders; dressings, bandages and shields for patients with ocular disorders; preparation for operation; instruments for operation on the eye, and the procedure of operation. The nursing care in cases of cataract is found in a chapter giving the history of the operation for cataract.

The directions for treatment are clearly outlined and will be useful to the ophthalmic nurse, particularly as the procedures enumerated are those that have stood the test of time in that excellent institution the Wills Hospital of Philadelphia.

ARNOLD KNAPP.

Medical Classics. Edited by E. C. Kelly, M.D., Albany Medical College, Albany, N. Y. In volumes of 10 numbers. Price, $10 per volume. Pp. about 1,000. Baltimore: Williams & Wilkins Company, 1936.

This publication aims to awaken the interest of all medical workers in the historical side of their profession. The important papers of previous great masters of medicine are printed in full, and, in addition, a brief life of the author, a bibliography, a brief sketch of the subject treated and the best available portrait of the author are given.

The subject chosen for the first number is Sir James Paget and his papers on osteitis deformans, Paget's disease of the bones, and his paper on Paget's disease of the nipple.

The subjects for the succeeding numbers are excellently chosen, and the publication deserves an enthusiastic reception. The importance of reading the original papers of the great masters cannot be overemphasized.

"Medical Classics" is dedicated to Dr. Joseph Lewi Donhauser. The editor welcomes suggestions concerning future subjects.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81, Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.
Time: June 11, 1937.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. William L. Benedict, 102 Second Ave., S. W., Rochester, Minn.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City. Time: June 7-11, 1937.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 3-5, 1937.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City, Utah. Time: May 24-27, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. H. Pember, 500 W. Milwaukee St., Janesville, Wis.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Building, Atlanta, Ga.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St. New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. E. C. Reynolds, 657 Main Ave., Passaic, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Carl T. Eber, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.
Place: Oscar Johnson Institute Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W. Toronto.
Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.